A COMPARATIVE STUDY OF VARIOUS PROGNOSTICS SCORING SYSTEM (RANSON, BISAP, CTSI) IN ACUTE PANCREATITIS

Dissertation submitted in

Partial fulfilment of the regulations required for the award of

M.S. DEGREE

In

GENERAL SURGERY – BRANCH - I

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL- 2013.

CERTIFICATE

This is to certify that the enclosed work **“" A COMPARATIVE STUDY OF VARIOUS PROGNOSTICS SCORING SYSTEM(RANSON, BISAP,CISI) IN ACUTE PANCREATITIS”** submitted by Dr V SARAVANA RAJA to The Tamilnadu Dr.M.G.R Medical University is based on bonafide cases studied and analyzed by the candidate in the Department of General Surgery, Coimbatore Medical College Hospital during the period of September 2011 – November 2012 under the guidance and supervision of **Professor D.N. Renganathan. M.S.(** General Surgery) and the conclusions reached in this study are his own.

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DECLARATION

I solemnly declare that the dissertation titled "**A COMPARATIVE STUDY OF VARIOUS PROGNOSTICS SCORING SYSTEM(RANSON, BISAP,CISI) IN ACUTE PANCREATITIS"** was done by me from September 2011 to November 2012 under the guidance and supervision of **Professor D.N. Renganathan M.S.(** General Surgery), Coimbatore Medical College, Coimbatore.

This dissertation is submitted to the Tamilnadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of MS Degree in General Surgery (Branch I)

I have not submitted this dissertation on any previous occasion to any University for the award of any degree

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Above all, I would like to express my heartfelt gratitude to all the patients, for their participation and ineffable cooperation during this study.

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LIST OF ABBREVATION USED

1. BISAP- bedside index of severity in acute pancreatitis
2. CTSI-CT severity index
3. SAP-severe acute pancreatitis
4. PAN NEC-pancreatic necrosis
5. CEA-carcinoembryonic antigen
6. ROC-Receiver operator curve
7. AUC-Area under curve
8. NPV-Negative predictive value
9. PPV-Positive predictive value
10. SD-Standard deviation
11. NP-Not prevalent
12. CECT-Contrast enhanced ct scan
13. SEN.-Sensitivity

A COMPARATIVE STUDY OF VARIOUS PROGNOSTICS SCORING SYSTEM (RANSON, BISAP, CTSI) IN ACUTE PANCREATITIS

ABSTRACT

Acute pancreatitis is a sudden inflammation of the pancreas due to many causes. There are many prognostic scoring systems to predict the severity and the outcome of the disease. The aim of the study is to compare the three scoring system namely RANSON, BISAP and CTSI in predicting the outcome such as acute severe pancreatitis, pancreatic necrosis, mortality and number of days stayed in hospital are considered in the study and to classify the pancreatitis according to atlanda classification and study its prognostic significance.

METHODS:

Extensive data from consecutive patients with AP admitted o to our Coimbatore Medical college was collected between September 2011 and November 2012. The BISAP scores were calculated using data from the first 24 h from admission and the RANSON score within 48 h from admission. Predictive accuracy of the scoring systems was measured by the area under the receiver-operating curve (AUC).

RESULTS:

There were 117 patients with AP (mean age 39+/-11). Incidence in males 94%. 21% patients developed organ failure and were classified as severe AP . 14%developed, and 7 died (mortality 5.9%). AUCs for BISAP in predicting SAP is 0.773 and predicting the mortality is 0.789. The AUC FOR CTSI on predicting the pancreatic necrosis is 0.814.

CONCLUSION

To conclude BISAP score is simple and it is the better scoring system in predicting the prognosis when compared to other score. The BISAP score has many advantages when compared to other scoring system. The CTSI score predicted the pancreatic necrosis well. Atlanta classification also holds good for the classify the pancreatitis into mild and severe disease also holds good to assess the prognosis of the acute pancreatitis.

KEY WORDS: acute pancreatitis, RANSON, CTSI, BISAP.

INTRODUCTION

Acute pancreatitis is a sudden inflammation of the pancreas due to many causes. The disease can range from mild to severe disease. More often mild disease can be treated with conservative management like nil by mouth and the pain which is severe, is managed. But severe case is bound to have many complications and they need a careful monitoring. We need to know the prognosis of the patients with severe disease so complications can be anticipated and patients who need monitoring can be found out.

There are many prognostic scoring systems to predict the severity and the outcome of the disease. In this study three scoring system namely RANSON, BISAP and CTSI are compared in predicting the outcome of acute pancreatitis. The outcome such as acute severe pancreatitis, pancreatic necrosis, mortality and number of days stayed in hospital are considered in the study. Then the scoring system are compared in predicting the mortality, pancreatic necrosis, acute severe pancreatitis.

CONCLUSION

To conclude BISAP score is simple and it is the better scoring system in predicting the prognosis when compared to other score. The BISAP score has many advantages when compared to other scoring system. The CTSI score predicted the pancreatic necrosis well. Atlanta classification also holds good for the classify the pancreatitis into mild and severe disease also holds good to assess the prognosis of the acute pancreatitis.

KEY WORDS: acute pancreatitis, RANSON, CTSI, BISAP.

AIMS OF THE STUDY

1) TO COMPARE THE SENSITIVITY, SPECIFICITY, POSITIVE AND NEGATIVE PREDICTIVE VALUE OF VARIOUS SCORING SYSTEM NAMELY RANSON SCORE, CTSI, BISAP IN PREDICTING THE SEVERE ACUTE PANCREATITIS, PANCREATIC NECROSIS, MORTALITY AND TO SUGGEST THE BEST SCORING SYSTEM AMONG THE THREE PROGNOSTIC INDICATER APPLICABLE TO OUR POPULATION

2) TO CLASSIFY THE PANCREATITIS ACCORDING TO ATLANDA CLASSIFICATION AND TO STUDY ITS PROGNOSTIC IMPORTANCE.

REVIEW OF LITERATURE

HISTORICAL REVIEW

- In Greek Pan means "All" and Kreas means "Flesh" and it was described first by Herophilus Chalcedon.
- Rufees and Ephesees-named the pancreas.
- Wirsung- described the main pancreatic duct.
- Santorini- illustrated the accessory duct bearing his name.
- Ringor de Graaf- the first reference to pancreatic lithiasis in 1664.
- Reiddle- chronic pancreatitis was first described by him.
- Kini- reported the first case of pancreatic calculi In India.
- Elizabeth and Stephen- reported 9 cases of pancreatic calculi from vellore.
- Chuttani and Anand- reported 32 cases of pancreatits from North India with gall bladder disease in 25% of cases and alcoholism in 6%.
- Brocks and Gifferd- performed the first human homotrans plant in 1959, using fragmented pancreatic tissue.
- Lillehei and colleagues- performed the first human pancreatic whole organ transplant in 1967.
- Ballinger and Lacy- popularised the concept of islet cell transplantation in 1972.

ANATOMY OF THE PANCREAS[1,2]

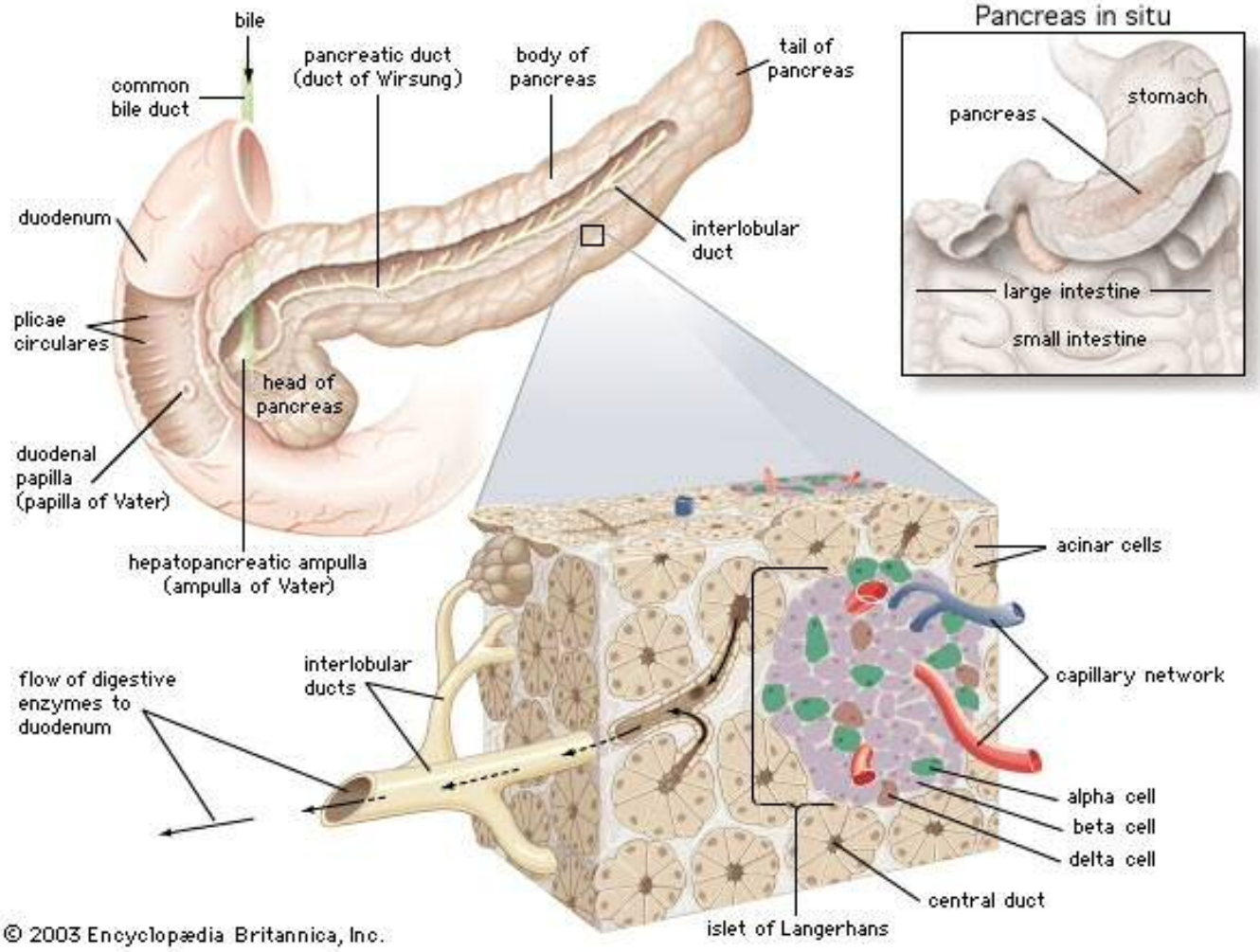
Pancreas is a retroperitoneal organ and it lies behind the stomach, transverse colon, and mesocolon. The whole, organ measures over 15 cm long weighs about 90 to 120 g in adult, soft in consistency with lobulated surface. It occupies the supracolic and partly the infracolic compartment. It comprises of head, neck, body and tail.

Head:

It is the broadest part, occupies the concavity of the duodenum, and lies over the inferior venacava, right and left renal veins. Its posterior surface is indented by last part of common bile duct. The uncinate process is the wedge shaped lower part of the gland, lies posterior to the superior mesenteric artery and vein and lies anterior to the aorta, at the level of L2.

Neck:

It is the continuation of the upper part of the head, lies anterior to the superior mesenteric vein and portal vein formation. It lies at the level of LI vertebra.


bile
common bile duct
pancreatic duct (duct of Wirsung)
body of pancreas
tail of pancreas
duodenum
interlobular duct
plicae circulares
head of pancreas
duodenal papilla (papilla of Vater)
hepatopancreatic ampulla (ampulla of Vater)
Pancreas in situ
stomach
pancreas
large intestine
small intestine
acinar cells
capillary network
interlobular ducts
flow of digestive enzymes to duodenum
alpha cell
beta cell
delta cell
central duct
islet of Langerhans
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Body:

The body starts from the neck and it runs across the left renal vein, left crus of the diaphragm, aorta, left psoas muscle, hilum or the left kidney and the lower pole of the left suprarenal gland. The Splenic artery passes along the upper border of the body and tail and it is tortuous in course. Splenic vein lies closely applied to its posterior surface. The Splenic vein gets its tributary from the inferior mesenteric vein behind the body of the pancreas. The transverse mesocolon is attached in the lower part of the anterior surface of the body and neck.

Tail:

It passes forwards from the anterior surface of the left kidney at the level of hilum, accompanied by splenic artery, splenic vein and lymphatic in the two layer of lieno renal ligament and then touches the hilum of the spleen.

Ductal system of pancreas:

The duct of Wirsung is the major duct comes from the tail to the head, arises from the confluence of numerous small ducts of the lobules crossing the gland forming a "Herring bone" pattern, gradually increasing in diameters upto 10 mm joins with the common bile duct in a dilatation, the ampulla of vater, which opens into the duodenal papilla. The accessory pancreatic duct drains the uncinate process and lower part of the head of pancreas lies more on ventral

plan, opens into the duodenum 2 cm proximal to the major papilla and 7 cm distal to the pylorus ". Injury to the duct of Santorini in the pancreatic divisum during gastrectomy results in severe hemorrhagic or recurrent pancreatitis.

Blood supply:

Blood supply is chiefly derived from splenic artery which supplies neck, body and tail by a large branch named as "arteria pancreatica magna". The head is supplied by superior pancreatico duodenal artery (a branch of coeliac artery) and inferior pancreatico duodenal artery (a branch of superior mesenteric artery). The right hepatic artery is a branch of superior mesenteric artery, passes behind the head of the pancreas or within its substance.

Venous drainage is by small veins into the splenic vein and the head of the pancreas drain into the superior pancreatico duodenal vein into the superior mesenteric vein which forms a landmark during pancreatic resection.

Lymphatic drainage:

Lymphatic drainage generally follows venous drainage in all directions. They drain into the following group of lymph nodes

A. Superior nodes drain the anterior and superior upper half of the gland.
B. Inferior nodes drain the anterior and posterior lower half.
C. Anterior nodes drain the anterior surface of the head of the pancreas.

D. Posterior nodes drain the posterior surface of the head.

E. Splenic nodes drain the tail of the pancreas.

Every group of lymph node finally drains into the coeliac and superior mesenteric group lymph nodes.

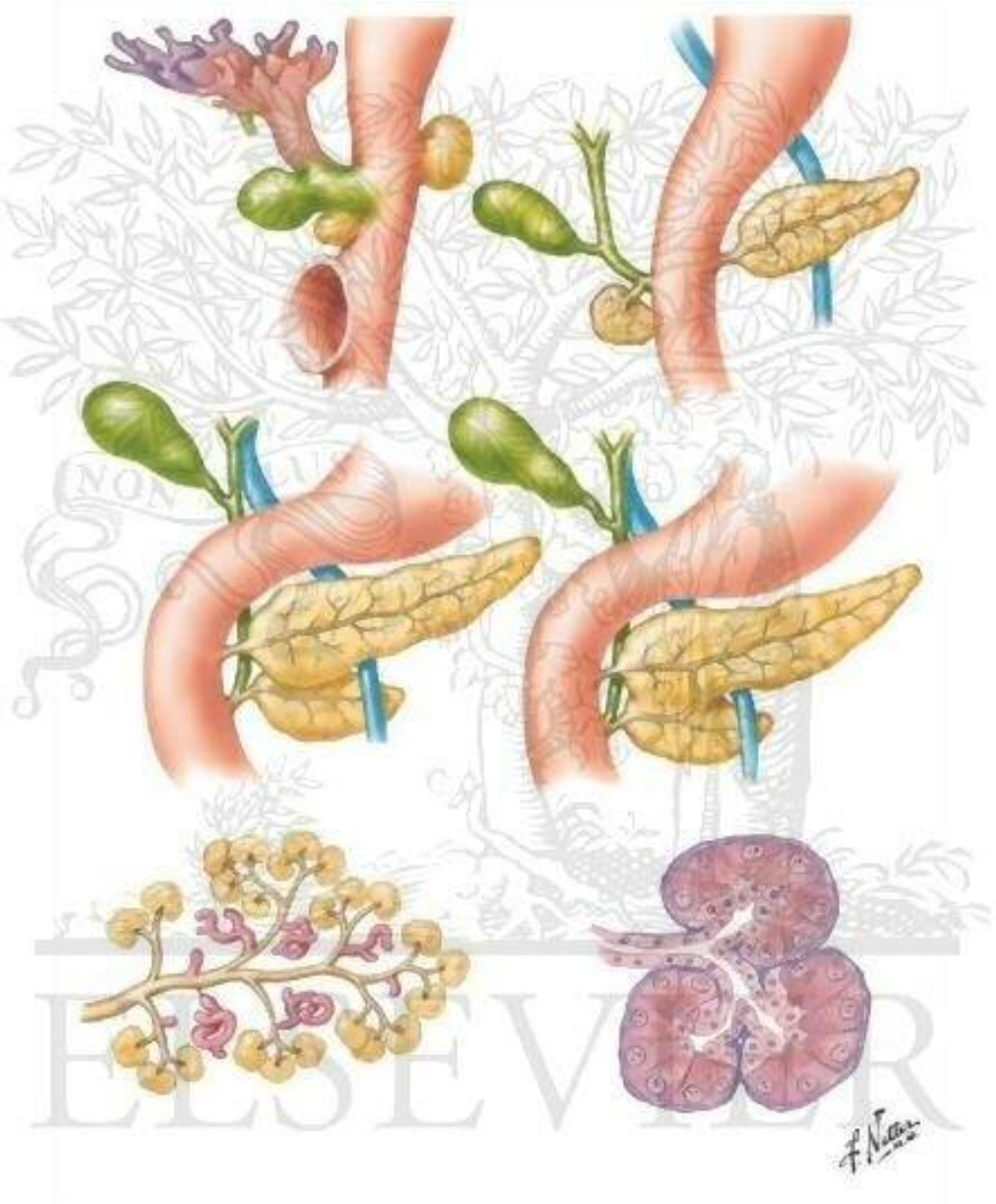
Nerve supply:

The afferent pain sensation from the pancreas is conducted through the sympathetic fibers from the greater, lesser and lowest splanchnic nerves via the central ganglia. The coeliac branch of the right vagus nerve provides the parasympathetic supply.

Development of pancreas:

Pancreas develops as two separate buds each an outgrowth of the endoderm at the junction of foregut and midget. The ventral bud grows into the ventral mesogastrium in common with the outgrowth of bile duct and the dorsal bud grows into the dorsal mesogastrium. The duodenal portion of the duct rotates and becomes adherent to the posterior abdominal wall, the ventral bud rotates and fuses with the dorsal bud at 7 to 8 weeks of gestation. The dorsal pancreatic duct by connecting with the ventral pancreatic duct becomes the major duct of Wirsung draining the body and tail, the proximal end is retained as accessory pancreatic duct of Santorini. This duct opens into the duodenum separately in 70 % of cases and in 5 % of cases it becomes the

major duct - pancreatic divisum.



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The pancreatic alveoli developed by the growth of cells from the terminal part of the branching ducts. The islet cells appear to have as identical origin but become separated from their parent ducts and undergo change of secretary function.

Microscopic anatomy:

This lobulated gland composed of alveoli of serous cells with, very few ducts without islet cells by characteristic staining reaction. In each alveolus the basal part of the cell is deeply stained and basophilic, while the central part is acidophilic. The nucleus is situated towards the basal part. The ducts are lined with simple columnar epithelium.

The islets, in section appear as pale areas, more prevalent in the tail with Zankes - formal fixation. It varies in size from one to four times that of pancreatic alveolus. Alpha cells produce glucagon which is situated more in the periphery of the islets, constitutes 18 to 25% of the cells. Beta cells producing insulin has secretory granules, density of which varies in patient to patient. The Delta cells producing somatostatin constitutes 3 to 8 % located near the alpha cells contains granules demonstrated by Electron Microscopy.

PHYSIOLOGY[18,4]

The pancreas has both the endocrine and exocrine functions. Exocrine pancreas - The acinar cells of the pancreas secrete enzymes and small amount of electrolytes. The centroacinar and ductular cells secrete water and electrolytes.

Composition - Total volume - 1500 to 2000 ml/day

Protein - 5to8 G pH - alkaline (8.3)

It is isoosmotic and alkalinity is due to the bicarbonate concentration which depends on the secretory rate (100 to 150 mmol/1). The Na^+ and K^+ concentration is similar to the plasma but other anions and chlorides are inversely related to the bicarbonate concentration and are flow dependant.

Proteins in pancreatic juice:

1. Amylolytic enzymes -alpha amylase
2. Proteolytic enzymes
   a)Endopeptidases-Chymotrypsinogen,Trypsinogen,Proelastases
   b)Exopeptidases- Procarboxypeptidase
3. lipolytic enzymes - lipase

4. Other enzymes – Phospholipase A, carbonyl ester hydrolase, Ribonuclease Deoxyribonuclease

5. Other proteins-Immuno globulins, Lactoferrin, CEA

REGULATION OF SECRETION

Both nervous and hormonal control.

I. Cephalic Phase

Stimuli similar to gastric secretion.

Efferent fibres - Vagus nerve

Volume of secretion - small

Enzyme - High

Hormone - Gastrin from antrum.

II. Gastric Phase

Secretion further stimulated.

Both nervous and humoral control

Distension of body of stomach excite tension in the wall Vasovagal reflex and causes increased enzyme output Gastrin release due to chemical or mechanical stimuli which produces enzyme rich sma11 volume secretion.

III. Intestinal Phase

Acid chyme enters the duodenum and causes the release of hormone secretin from the endocrine cells of the mucosa. Secretin stimulates watery secretion and an isoosmotic solution of bicarbonates.

Pancreozymin hormone from the I cells in crypts and villi of duodenum and jejunum on release stimulates enzyme rich secretions.

ACUTE PANCREATITIS

Defined as pancreatic inflammation followed by clinical and biological restitution gland if the primary cause is eliminated. Different stages are distinguished in the development of acute pancreatitis. There are a number of known and unknown etiologic factors capable of initiating pancreatic inflammation in a variety of ways that finally results in pancreatic necrosis

Pancreatic involvement may be confined to the initial damage and may cease spontaneously or gives rise to an activation of digestive enzymes within the pancreas thereby self perpetuating pancreatic auto digestion with fat necrosis and hemorrhage.

ETIOLOGIC FACTORS[2]

A number of factors either acting alone or a combination of them may be responsible for the pancreatic onslaught, Etiological factors in acute pancreatitis

I. METOB0LIC

- A1cohol
- Hyper1ipoprotenemia
- Hypercalcemia
- Drugs
- Scorpion venom

II MECHANICAL

- Choielithiasis
- Post, operative [gastric, biliary]
- Post traumatic
- Obstruction of the duct
- Pancreatic tumor
- Duodual obstruction

III VASCULAR

- Post operative (cardio pulmonary bypass)
- Periarteitis nodosa
- Atheroembolism

IV INFECTIONS

- Mumps
- Coxsackies Infection

DEVELOPMENT OF ACUTE PANCREATITIS[4,5]

Etiological factor described above initiate the process of bile reflux and causes the pancreatic injury. The pancreatic injury is manifested as edema, vascular injury, and pancreatic acinar damage. This injury causes the activation of the pancreatic enzymes such as trypsin, phospholipase A etc. This leads to autodigestion and pancreatic necrosis.

MECHANISM BY WHICH COMMEN ETIOLOGICAL FACTOR CAUSES ACUTE PANCREATITIS

A) ALCOHOL - MECHANISM OF INJURY

- Panacreatic exocrine hypersecretion in the presence of partial

ampullary obstruction.

1. Alcohol is a stimulant of gastric acid sceretion, and the resultant duodenal acidification relases secretin which increases the exocrine pancreatic secretion of water and bicarbonate
2. Alcohol also increases the resistance of sphincter of Oddi causing partial obstruction to the flow of pancreatic secret ion.
3. Alcohol increases the intraductal pressure in pancreatic ducts and also increases permeability of ducts to macromolecules.

- Alcohol initiates enzyme extravasation and cause pancreatic injury as a result of protein obstruction of the pancreatic duct.
- Intermediate state of hypertriglyceredemie following alcohol ingestion. Toxic levels of free fatty acids, produced from the lipolysis of triglycerides may cause acinar cell or capillary endothelial cell injury in the pancreas.

B) GALL STONES

Mechanism

Gall stone migration through the ampulla of Vater, causes diversion of bile into the pancreatic duct and subsequent bile-induced pancreatic parenchymal injury.

Evidences

- Presence of Gall stones in stools of 90% of patients with acute Gall stone pancreatitis.
- Cholangiographic studies show a common channel between the CBD and pancreatic duct in 90% of patients with Gallstone pancreatitis.
- Intraoperative cholangiogram after cholecystectomy shows that pancreatic duct reflux in 60% of patients with history of pancreatitis.
- Endoscopic recovery of stones impacted at Ampulle of Vater within 48hrs of onset of symptoms.

C) HYPERLIPOPROTENEMIA

D) Pancreatitis associated with various primary hyperlipoprotenemic conditions are as follows.

Disease		% occurance of pancreatitis
Fredrickson	Type I	30%
	Type IV	15%
	Type V	27 - 41%

Since Type IV is the commonest form of Hyperlipoprotenemia, this accounts for mo6t examples of lipid associated pancreatitis. Free fatty acids released by pancreatic lipase may exert a toxic influence as the pancreatic parenchyma.

E) HYPERPARATHYROIDISM AND HYPERCALCEMIA

Incidence-7-19%

Mechanism

- Calcium induced trypsinogen activation and subsequent auto destruction.
- Calcium associated stone precipitation in the duct causing obstruction.
- Calcium stimulated pancreatic exocrine hypersecretion.

Direct toxic effect on parenchyma of pancreas by paratharmone.

MECHANISM OF ACUTE PANCREATITIS[4,5]

I. INTRA PANCREATIC ACTIVATION OF PANCREATIC ZYMOGENS

The cardinal mechanism is the activation of the trypinogen to trypsin and this enzyme activates the other enzymes and the pathology continues. Whatever may be the etiology finally it lands upon the above given mechanism.

A concept known as the intrapancreatic activation of the enzymes in postulated. The release of the pancreatic enzyme is hindered and they join the intracellular lysosomes and this results in the activation of the proenzyme trypinogen to trypsin. This results in activating all known pancreatic zymogens like chymotrypsinogen to active chymotrypsin, proelastase to elastase and prophospholipase to lipase A. Only lipase already synthesized in active form is independent of trypsin. Every activated enzyme has its own function and it is summarized in the flow chart.

Of the all etiological factor alcohol is the most common cause of the acute pancreatitis so its mechanism is discussed. The mechanism is as follows.

- Hyper secretion of the exocrine pancreatic secretion in the presence of the partial ampullary obstruction.
- Enzyme extravasations initiating the pancreatic injury
- Alcoholic usually have hypertrigiyceredemia this also initiates the pancreatitis.

The next common cause in the gallstone pancreatitis. Gall stone migrates into the ampulla of vater which causes the diversion of the bile into the pancreatic duct which results in the bile induced pancreatic injury.

PATHOLOGICAL CHANGES ACUTE PANCREATITIS[5]

Mildest pathological change - Edema of the gland. May be accompanied by infiltration of the intralobular septa by inflammatory cells.

Microscopy-fat necrosis in the pancreas and surrounding tissues.

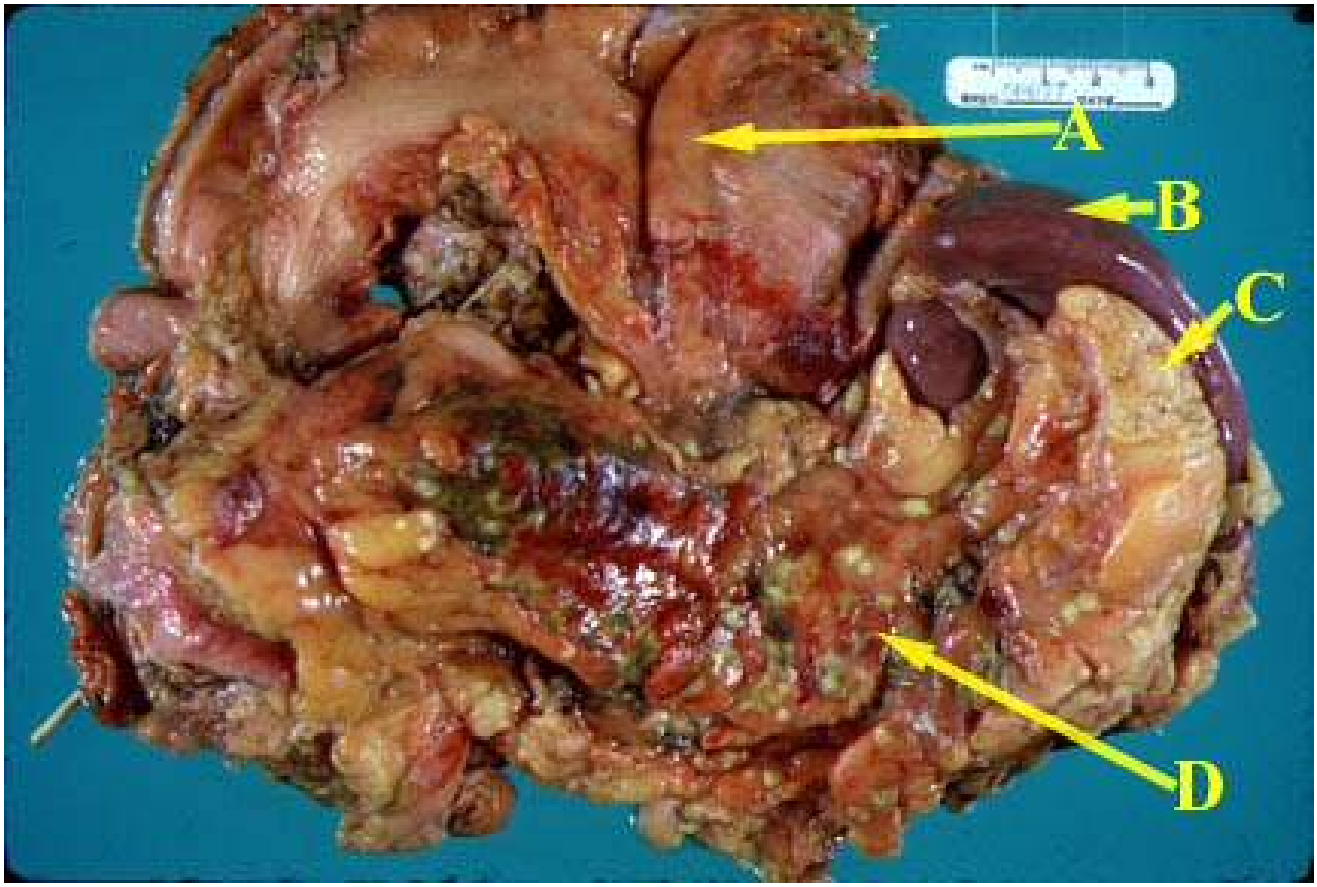
If extensive necrosis - Whitish yellow plaques occur due to necrosis and calcium deposition.

Vascular thrombosis or disruption results in pancreatic necrosis or gross hemorrhagic infarction.

Increased levels of active pancreatic enzymes occur,

1. within pancreas
2. in the peritoneal exudate
3. in the blood stream of patients with pancreatitis

FIG 4 ACUTE PANCREATITIS SPECIMEN



The specimen shows acute pancreatitis with severe necrosis

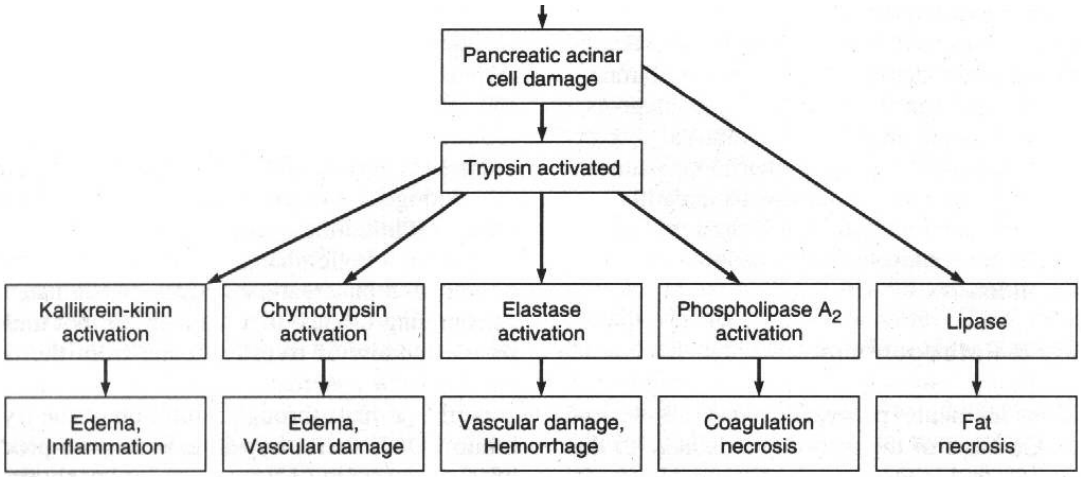


Figure 15–3. Hypothesized pathogenesis of acute pancreatitis. (Reproduced with permission from Marshall JB: Acute pancreatitis: A review with an emphasis on new developments. Arch Intern Med 1993;153:1185.)

CHANGES IN DIFFRENT SYSTEM IN PANCREATITIS

The pathophysiology alters many system in the body. The changes affects the following system.

- Fluid and electrolyte changes
- Cardiovascular changes
- Respiratory changes
- Renal changes
- Local changes

I. FLUID AND ELECTROLYTE CHANGES

Circulating blood volume decreased due to loss from intravascular space of plasma into the retroperitoneum and systemically. Additional loss occur following vomiting or naso gastric aspiration. Hypocalcaemia and hypomagnesemia are frequent. Decreased ionised calcium level also occurs due to trapping of calcium in areas of fat necrosis.

II. CARDIOVASCULAR CHANGES

Hypotension, Tachycardia, increased total perpheral resistance and decreased cardiac output - sequelae of hypovolemia also observed in acute pancreatitis similar to septic shock or Hepatic cirrhosis are due to circulatory vasoactive substances. Hypotension persists despite

restoration of intravascular volume.

III. RESPIRATORY COMPLICATIONS

- Early feature of acute pancreatitis -arterial hypoxemia
- Pulmonary function studies - Decreased inspiratory lung volume with decreased pulmonary compliance and decreased diffusing capacity.
- Early respiratory failure resolves with subsidence of pancreatitis.
- Severe or unresolving pancreatitis may develop progressive pulmonary insufficiency, infiltrates and pleural effusion.

IV. FACTOR IMPLICATED FOR PULMONARY COMPLICATIONS

1. Abdominal distension and elevation of diaphragm.
2. Alteration in the lecithin of pulmonary surfactant by circulating pancreatic lecithinase.
3. Pulmonary thromboembolism.
4. Circulating free fatty acids
5. Circulating products of the proteolytic cleavge complement.

V. RENAL FAILURE

Major factor is deaths from pancreatitis. Due principally to hypovolemic. So many patients go in for acute renal failure. Pathologically it is due to the deposition of the fibrin complexes in the glomeruli.

VI. OTHER SYSTEMIC FEATURES

Abnormal Liver functions-Elevation of serum bilirubin and liver enzymes such as Alkaline Phosphatase are raised and it is mainly due to the biliary obstruction and pericholangitis.

Early Intravascular thrombosis with decreased platelet count and fibrinogen level occur due to the effects of pancreatic proleolytic enzymes. May be followed by marked thrombocytosis and hyperfibrinogenemia.

VII. LOCAL SEQULAE

Intra abdominal complications

1) Paralytic ileus
2) Duodenal/Biliary obstruction
3) Release of pancreatic enzymes with peripancreatic fluid collection and fluid in general peritoneal cavity.

4) Destruction of tissues adjacent to pancreas.
5) Rarely cause gross disruption of the pancreatic ductal system which is usually self limited.
6) Persistent chronic pseudocyst in 1%
7) Infected pancreatic abscess due to secondary infection occur in 1-9% and organisms are usually enteric.
8) Extension of local necrosis to involve colonic wall causing colonic perforation occurs in 1% and occurs in the left transverse colon or splenic flexure.

CLINICAL MANIFESTATIONS AND DIAGNOSIS[2,3,10]

The classical feature of acute pancreatitis is its severity of symptoms and paucity of physical signs.

1. Abdominal pain - 85 -100%

Upper abdominal constant pain may radiate to the back and-may be severe. Pain is aggravated by the food or by a drink of alcohol. Pain resistant to analgesics. Patient assumues of various postures in an effort to obtain relief.

2. Nausea and vomiting - 92%

Vomiting is usually non projectile and it is of low volume and it

contains gastric and duodenal content and it is not feculent.

3. Physical examination

- Restless patient.
- Rapid pulse and respiratory rate.
- Arterial hypotension
- Abdomen- moderately distended with epigastric dullness Tenderness markedly in the upper abdomen.
- Moderate muscle spasm present.
- **GREY TURNERS SIGN** - Grey green discoloration of the flank in patients with peripancreatic heamorrhage
- **CULLEN' S SIGN** - bluish discoloration of periumbilical region

4. Extra abdominal manifestations

- Left pleural effusion
- Acute pulmonary failure marked by Tachypnoea, dyspnoea,
- cyanosis - due to
    a) circulating phospholipase A
    b) circulating free fatty acids from triglycerides from lipolysis
    c) Pulmonary surfactant
    d) Volume overload with pulmonary capillary leakage

5. Central Nervous System manifestations-

Nonlateralizing nature, including billigerence, confusion, psychosis and coma. This is due to hyperosmolarity, hypoperfusion, hypoxia, cerebral fat embolism or Disseminated intra vascular coagulation.

LABORATORY DETERMINATION[2,18]

DIAGNOSIS OF ACUTE PANCREATITIS

Laboratory Tests	Radiographic Procedures
• Sr. amylase	• Chest X Ray
• Sr. amylase isoenzymes	• Plain abdominal X Ray
• Urine amylase	• Ultrasonography
• Amylase-creatinine clearance ratio	• Contrast enhanced C T SCAN

I. BLOOD COUNT :

- Leucocytosis - 10,000 to 20,000 occurs early in all cases
- Haematocrit - is high in most patients at the onset.
- Haemoglobin decreased value of more than 2.5G% without detectable blood loss found in those with pancreatic necrosis

II. SERUM AMYLASE

Elevated in 95% of patients with Acute pancreatitis. But this is not an ideal marker because it is elevated in other conditions such as

A. Perforated peptic ulcer

B. Biliary lithiasis

C. Intestinal obstruction

D. Mesenteric infarction.

Also in patients with acute pancreatitis, serum amylase in normal levels can occur due to

I. Hyper triglyceredemia - Latescent serum

II. Assayed 3 days or more after onset

III. Previous attack has destroyed most glandular tissues

IV. Present attack is associated with massive destruction of gland

Serum amylase in Acute Pancreatitis is elevated within 24 hrs of onset of symptoms and returns to normal in 7 days.

III. SERUM ISOAMYLASE P:

As it is produced only from pancreas it has a higher specificity in detection and confirmation of acute pancreatitis.

IV. SERUM LIPASE:

Serum lipase is solely of pancreatic origin hence serum lipase level is more specific than amylase. Recent development of an enzyme immuno assay of lipase is reliable and is of great value in Acute Pancreatitis. Duration of Hyper lipasemia exceeds hyperamylossemia.

V. PLURAL AND PERITONEAL FLUID AMYLASE

Pleural effusion shows raised levels of amylolytic activity in pancreatitis. High activities of amylase may also be found in fluids aspirated from peritoneal cavity in patients with acute pancreatitis.

IV. OTHER BIOCHEMICAL INDICES

I. Hyperglycemia and Glycoscuia- Nonspecific, transient
   Cause - relative hypoinsulinemia and Hyperglucognaemia
II. Hypocalcemia - Well recognised entity in acute pancreatitis but can also occur in perforated peptic ulcer

Cause - Deposition of calcium in areas of fat necrosis Release of glucagon Inadequate parathyroid response Dilutional hypo albuminaemia

i. Methemalbumin:

Appearance in serum indicates necrotic rather than edematous pancreatitis.

ii. Liver function tests:

Slight increases in alkaline phosphatase and amino transfer are with raise in serum bilirubin - transitions. Markedly elevated serum aspartate and alanine amino transfer are within 48 hrs after onset discrimianates biliary from non-biliary pancreatitis.

RADIOGRAPHIC FINDINGS[2,18]

I PLAIN X-RAYS

i). Plain X-Ray abdomen

a) Segmental small bowel ileus or a "SENTINEL LOOP' in the left upper quadrant.

b) Dilatation of the transverse colon – "COLON CUT OFF SIGN".

A. Increase epigastric soft tissue density

B. Obscured psoas muscle margins.

C. Presence of gall stones

D. Pancreatic calcification – may not be an acute pancreatitis

ii). Plain x-ray chest

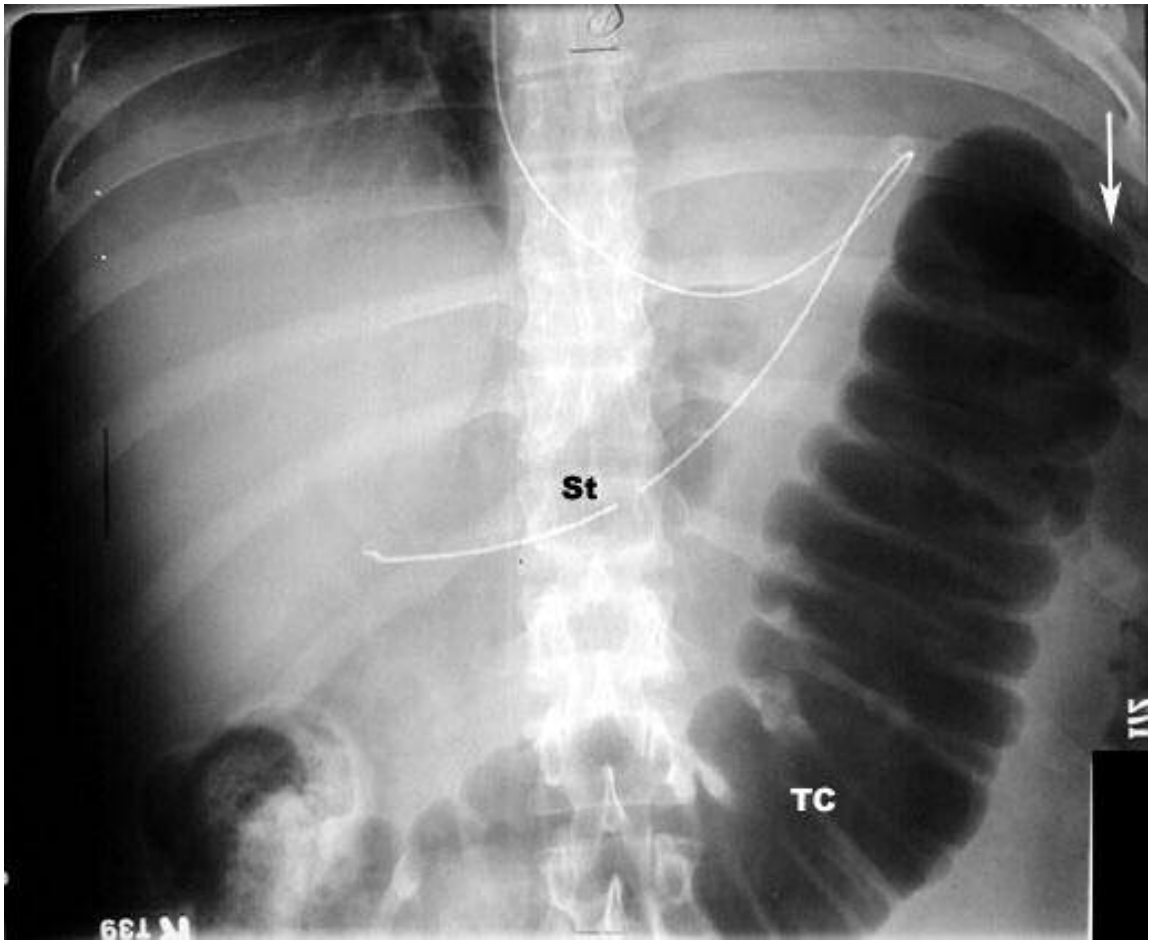
(a) Pleural effusion

(b) Atelectasis

(c) Pneumonia

(d) Pulmonary edema

COLON CUT OFF SIGN



CONTRAST STUDIES WITH WATER SOLUBLE CONTRAST

Upper Gastrointestinal study

(a) widening of "C" loop

(b) Anterior displacement of stomach

(c) Subtle duodenal mucosal sign

ABDOMINAL ULTRASONOGRAM

(a) enlargement and edema of pancreas

(b) Pseudocysts of pancreas

(c) Delineates pancreatic abscess

(d) Dilatation of Bile duct and presence of stone in gall bladder and common vile duct

COMPUTED TOMOGRAPHY

Except in early or mild eases it is useful in assessing Ct scan reveals many finding in the pancreatitis. Pancreas is usually enlarged and there is pancreatic edema. Pancreatic necrosis is characterized by the non enhancement in the contrast Ct scan. In the peri pancreatic area there is collection, obliteration of the fat plane and thickening of the fat plane. Other finding are ileus, pleural effusion.

VI ENDOSCOPY

To detect biliary pancreatitis.

Therapeutically used for papillotomy and removal of stones impacted at the ampulla of Vater.

TREATMENT[3,10]

MEDICAL MANAGEMENT

A. NUTRITION

1. Enteral nutrition

Previously it was thought that enteral nutrition stimulates the pancreas and results in pain in pancreatitis, but now it is found that pancreas is actually in a state of rest in acute pancreatitis so it better to stimulate the pancreas. So enternal feeding does no harm to the patient.

2. Total parenteral nutrition

TPN is associated with many complications such as arterial injury, pneumothorax, thrombosis, and catheter embolism. Many studies comfirm that enternal nutrition is better than TPN.

B. GASTRITIS PREVENTION

Patients suffering from severe AP have risk to develop peptic ulcers or erosive gastritis Histamin2-antagonists IS indicated in patient on mechanical ventilation and patients with adult respiratory distress syndrome (ARDS)

C. FLUID MANAGEMENT

Adequate fluid management is the main stay in the management of acute pancreatitis. If missed it can lead to serious complications. Plenty of fluids are sequestrated in third spaces. So crystalloids and colloids are used in the ratio of 3:1. The fluid loss may be 6-10 liters. It is said that when hematocrit is less than 30% if Dextran is used it improves the microcirculation. Good fluid resuscitation is indicated by adequate urine out put, CVP AROUND 8-12 cm of water, hematocrit of 35-40%.

D. PAIN MANAGEMENT

As a result of the activation of pancreatic enzymes there are increase releases of the inflammatory mediators. These mediators irritate the sensory fibers of the celiac plexus (T5-T9) and cause severe pain radiating to the back. The following drugs are used in management of pain

- nonsteroidal analgesia

- meperidine
- Tramadol
- thoracic epidural analgesia

E. THE ROLE OF ANTIBIOTICS[9]

There is a great controversy regarding use of antibiotic in acute pancreatitis. Since there is great risk in developing necrosis in acute pancreatitis, there is also problem in developing abscess in acute pancreatitis. So in order to prevent the abscess formation antibiotics are used. The most common antibiotics used are imipenem, meropenem, metronidazole, fluoroquinolones, and cephalosporins. These antibiotics penetrate the pancreas well, but aminoglycosids do not penetrate the pancreas. Over use of antibiotics result in the fungal infection.

F. SUPPRESSION OF PANCREATIC EXOCRINE SECRETION

These are done by nasogastric suction, histamine H_2-receptor antagonists, antacids,atropine, glucagon, calcitonin, stomatostatin.

SURGICAL MANAGEMENT: INDICATIONS AND TIMING

INDICATIONS[14,19,23]

1. Uncertainity of diagnosis

2. Treatment of pancreatic sepsis
3. Correction of associated biliary tract disease
4. Progressive clinical deterioration despite optimal supportive care.
5. Infected necrosis
6. Severe sterile necrosis
7. Symptomatic organized pancreatic necrosis

A) BILIARY OPERATIONS IN PATIENTS WITH CHOLELITHIASIS

a. Cholecystostomy
b. Common duct drainage
c. Cholecystectomy
d. Early endoscopic papillotomy

In patients with severe gall stone pancreatitis early intraabdominal surgery has been associated with higher mortality than early non operative treatment. Surgical correction of cholelithiasis to prevent recurrent pancreatitis undertaken once evidence of pancreattitis has subsided usually during the same hospital admission.

B) SURGICAL MANAGEMENT: PROCEDURES[19,23]

1. RESECTION
2. PANCREATIC DEBRIDEMENT
3. MINIMALLY INVASIVE APPROACHES
    - Retroperitoneal approach via dorsal lumbotomy
    - Percutaneous necrosectomy and sinus tract endoscopy

RESECTION

Pancreatic resection is primarily of historical interest only and is not recommended currently

PANCREATIC DEBRIDEMENT

All pancreatic débridement and postdébridement care are based on :

(1) Wide removal of devitalized and necrotic tissue

(2) the assurance of postoperative removal of the products of ongoing local inflammation and infection

TECHNIQUES OF DÉBRIDEMENT

1. Débridement and closed drainage

2. Open packing for pancreatic necrosis
3. Débridement And Continuous Closed Postoperative Lavage Of The Lesser Sac

THE COMPLICATIONS OF ACUTE PANCREATITIS[2,18]

The Complications of Acute Pancreatitis	
Local	Fluid collections
	Pancreatic ascites/pleural effusion
	Pancreatic pseudocyst
	Pancreatic necrosis
	Infected pancreatic abscess
	Hemorrhage/pseudoaneurysm
Regional	Venous thrombosis
	Paralytic ileus
	Intestinal obstruction
	Intestinal ischemia/necrosis
	Cholestasis
Systemic	Systemic inflammatory response syndrome
	Multiple-organ-dysfunction syndrome
	ARDS/pulmonary failure
	Renal failure
	Cardiovascular complications
	Hypocalcemia
	Hyperglycemia

	Disseminated intravascular coagulopathy
	Protein calorie malnutrition

A) PANCREATIC ABSCESS

Pancreatic abscess - Incidence '9%. Most common in patients with post operative pancreatitis.

Clinical features

Persistent of recurrent fever
Abdominal distension
Abdominal mass
Hypotension (BP 9 0 mmHg).
Pneumonia / Effusion
Renal failure
Coma
Elevated serum amylase
Leucocytosis (10000 /mm^)

Radio graphic diagnosis

1. Upper GI contrast studies showing di splacement of stomach or duodenum. Gas outside :GIT,.

2. Ultra sound abdomen can delineate pancreatic abscess
3. Computed Tomography sensitive and specif.lc
4. Percutaneous aspiration under CT guidance

Treatment

Adjuvant

1. Vigorous supportive management
2. Meticulous respiratory care nutritional support
3. Prevention of GIT haemorrhage

Specific

1. Percutaneous drainage by catheter
2. Laprotomy - Debridement and packing of the pancreatic bed.
3. Surgical correction of other complications like

involvement of colon by colostomy

4 Feeding jejunostomy to correct nutritional imbalance

B) PSEUDOCYSTS

Pseudocysts following acute pancreatitis spontaneous diSAPpearance of pseudocysts is a common occurence in acute pancreatitis. These are carefully monitored by serial ultrasonogram or CT and operative

intervention is needed only when they go in for further complications. It is dealt in detail along with treatment for pseudocysts following chronic pancreatitis.

C)**PANCREATIC ASCITES**

Pancreatic ascites - more common following chronic **pancreatitis**. But may also follow acute pancreatitis secondary to trauma, pseudocysts and rarely pancreatic neoplasm.Treatment is by drainage procedures as dealt in pancreatic ascites following chronic pancreatitis.

PROGNOSTIC ASSESSMENT

PROGNOSTIC INDICATORS

Because of the variability and unpredictability of acute pancreatitis, clinical scoring systems have been made to predict the severity of acute pancreatitis

1 . RANSON S CRITERIA[6]

RANSON criteria is the most commonly used scoring system and is based on 11 parameters measured within the first 48 hours of admission to the hospital .It is detailed in the tabular column

RANSON's Criteria

RANSON's Criteria	Nonbiliary Acute Pancreatitis	Biliary Acute Pancreatitis
Admission		
Age (yr)	>55	>70
WBC count (×1000/mm^3)	>16	>18
Glucose (mg/dl)	>200	>220
AST (IU/L)	>250	>250
LDH (IU/L)	>350	>400
Within 48 Hours of Admission		
Hematocrit decrease (points)	>10	>10
BUN increase (mg/dl)	>5	>2
Deficit in base(mEq/L)	>4	>5
Fluid replaced (L)	>6	>4
PaO_2 (mm Hg)	<60	<60
Calcium (mg/dl)	<8	<8

To calculate base deficit first bicarb VD must be calculated

Bicarb Vd =(0.4+2.6/HCO_3) x Lean Body Weight

Base Deficit= Bicarb Vd x (Normal HCO3- Measured $HCO_{3)}$

RANSON's score interpretation

According to RANSON score when the score is > 8 it indicates pancreatic necrosis upto 30% of the gland. If the score is >= to three the severe pancreatitis is likely and if it is < than three the severity is unlikely.

Score 0 to 2: The mortality is 2%

Score 3 to 4: The mortality is 17%

Score 5 to 6: The mortality is 45%

Score 7 to 8: The mortality is nears 100%

In this study the cut of value of the score is 3.the patients are classified under two groups one with RANSON score < 3 another with score >=3 since a score less than three severe pancreatitis is unlikely.

2. BISAP SCORE[13]

The BISAP is the simplest score in identifying the patients with rule of

mortality and severity in 24hrs period calculation of risk can improve the clinical care. The score was developed in state of pennyyslvania using a huge cardinal health database.

INDIVIDUAL COMPONENTS OF THE BISAP SCORING SYSTEM

- Impaired conscious level (GCSS < 15)
- Blood urea level > 25mg/dl
- Age > 60
- Pleural effusion
- Systemic inflammatory response syndrome

  The syndrome contains two or more of the following :

  (1) Temperature of < 36 or > 38 ° C

  (2) Respiratory rate > 20 breaths/min or P a CO2 < 32 mm Hg

  (3) Pulse > 90 beats/min

  (4) WBC < 4,000 or >12,000 cells/mm 3 or >10% immature bands

The total score is 5. In this study the cut of value of the score is 3, the patient are classified into two group one group with score < 3 and the other group with score >= 3.

3) CT SEVERITY INDEX[2]

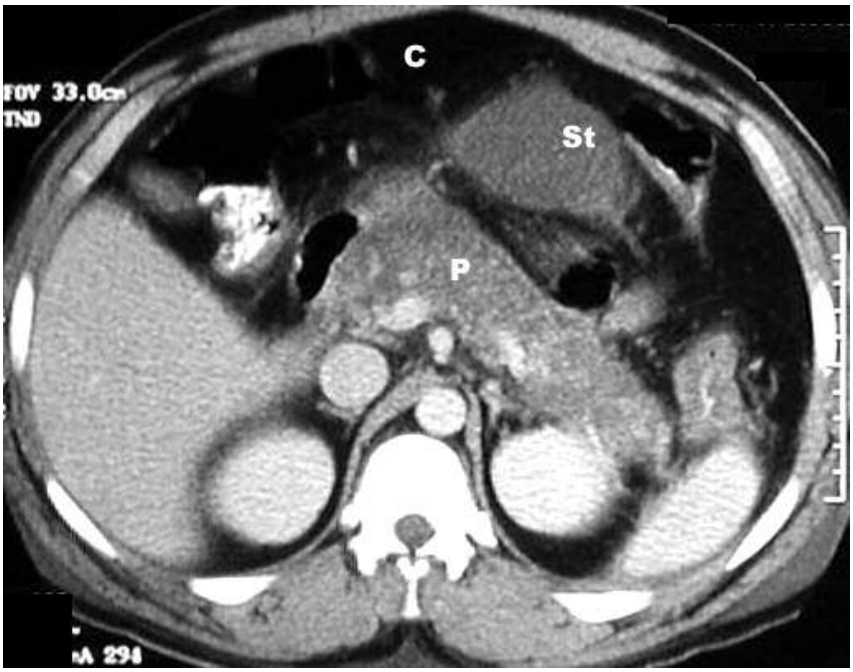
This uses CT scan primarily to grade the severity. CT scan is used to find the

pancreatic changes and necrosis. Either contrast or plain ct is used. This is developed by BALTHAZAR and AP is graded from A to E. It gives point according to the following criteria,

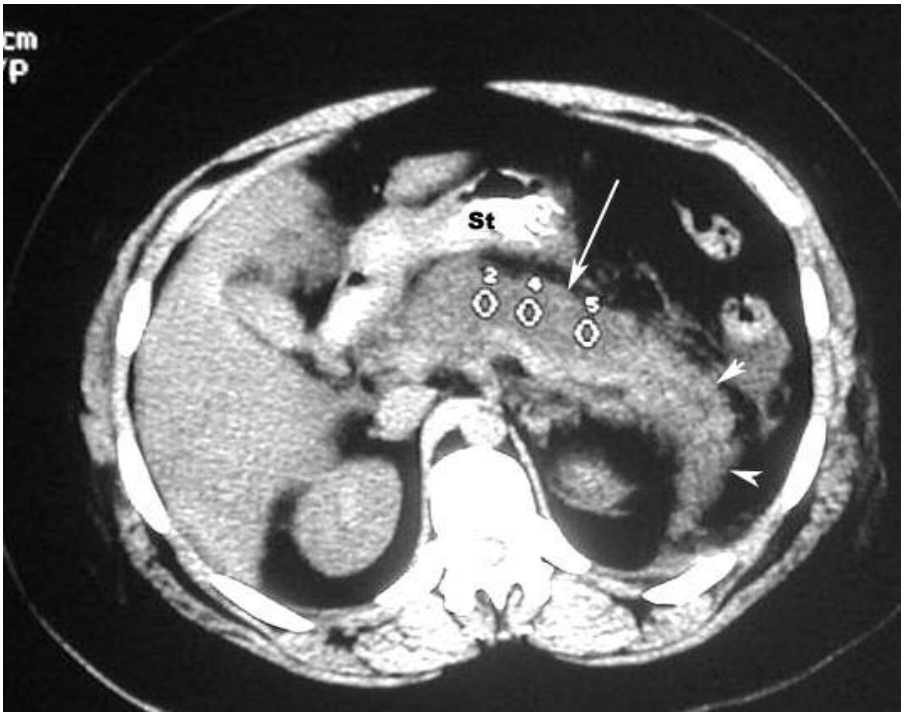
1. Nature of necrosis

2. peri pancreatic changes

ACUTE PANCREATITIS WITH EDEMA



SEVERE NECROTIC PANCREATITIS



Mild pancreatitis, interstitial pancreatitis

Patients with pancreatitis having no collections or necrosis. They have a mild pancreatitis. Balthazar grade A-C come under this group. CTSI is 2

Severe pancreatitis or necrotizing pancreatitis

They occur in 20 of patients. They have uneven clinical course and high mortality rate. They are more than fluid collections. The grade is d or e and this score is usually above 3. The peripancreatic collection is due to fat necrosis. This group of patients with. Necrosis has most complication and they have to be identified. There is a separate type called extra pancreatic necrosis which has on pancreatic necrosis the CTSI is 4.

Balthazar Scoring for the Grading of Acute Pancreatitis

Grade A – normal CT

Grade B – focal or diffuse enlargement of the pancreas

Grade C – pancreatic gland abnormalities and peripancreatic inflammation

Grade D – fluid collection in a single location

Grade E – two or more collections and/or gas bubbles in or adjacent to pancreas

CT severity index = CT grade point + points for necrosis

First grade points are calculated. The grade A, B, C, D, E have 0, 1, 2, 3, 4 respectively. The points for necrosis are based on the percentage of

necrosis. When the necrosis is <30% the point is 2, 30-50% the point is 4,>50% the point is 6.

CT grade points are added to points assigned for percentage of necrosis to determine the CT severity index. So the patients with score greater than three is said to manifest severe disease. So in this study two category one with score < 3 and another with score > =3 are taken into consideration

OTHER PROGNOSISTIC SYSTEMS

- The Acute Physiology and Chronic Health Evaluation II (APACHE II) score.
- C-reactive protein (CRP) assays.
- Trypsinogen-activating peptide (TAP) assays.

ATLANDA CLASSIFICATION

1) ACUTE ODEMATOUS PANCREATITIS – MILDER FORM MORTALITY 1%
2) ACUTE NECROTISING PANCREATITIS – incidence of 20% and characterized by pancreatic necrosis. The mortality is 15-20%.

MATERIALS AND METHOD

STUDY AREA :

GOVT. COIMBATORE MEDICAL COLLEGE AND HOSPITAL

STUDY POPULATION :

Patients admitted in CMCH with symptoms suggestive of acute pancreatitis

INCLUSION CRITERIA :

1. Clinical findings suggestive of abdominal pain characteristic of acute pancreatitis, serum amylase raised 3 times above normal value, ct scan with finding suggestive of acute pancreatitis data from the patient admitted with acute pancreatitis was collated. When some values are missed the referred case no mark is given. The data for RANSON is collected for 24 hrs and 48 hrs. CT scan is taken within 48 hrs. BISAP is calculated with 24 hrs data.

The Following criteria are studied

1. Severe pancreatitis
2. Pancreatic necrosis
3. Morbidity
4. Mortality

DEFINITION

1. MILD OR SEVERE AP

Depending on the organ failure the patients are classified into mild or severe pancreatitis. The presence of organ failure is again dictated by the presence of following factor

- Pulmonary failure(pco2 < 60mm of hg)
- Renal failure(serum creatinine levels > 2 mg/dl)
- Severe shock

2. PANCREATIC NECROSIS

This finding is easily found by the CECT scan. This is detected by the no enhancement in the CECT scan in the pancreatic parenchyma.

3. MORBIDITITY

Length of the hospital is used as the indicator of the morbidity

EXCLUSION CRITERIA :

1. Pediatric patients were excluded from the study.
2. Pregnant and postpartum patients were excluded

STUDY PERIOD:

September 2011, November 2012.

SAMPLE SIZE:

All patients eligible by inclusion and exclusion criteria are to be included in the study.

STUDY DESIGN:

A cross sectional observational study

RESULTS

STATISTICAL ANALYSIS:

The data are reported as the mean +/- SD or the median, depending on their distribution. The differences in quantitative variables between groups were assessed by means of the Unpaired t test. Comparison between groups was made by the Non parametric Mann - Whitney test. The chi square test was used assess differences in categorical variables between groups. Data were analyzed by diagnostic efficiency derived from the receiver operating characteristic [ROC] Curve and area under the ROC curve. Sensitivity, specificity and predictive values were determined. A p value of <0.05 using a two-tailed test was taken as being of significance for all statistical tests. All data were analyzed with a statistical software package. (SPSS, version 16.0 for windows)

After calculating the sensitivity, specificity, PPV, NPV for the scoring system such as RANSON, BISAP,CTSI in predicting the mortality, pancreatic necrosis, acute severe pancreatitis, the results are compared to derive at the conclusion.

The results are summarized with the explanation below

A) AGE SEX DISTRIBUTION

Table 1 – Age Sex Distribution

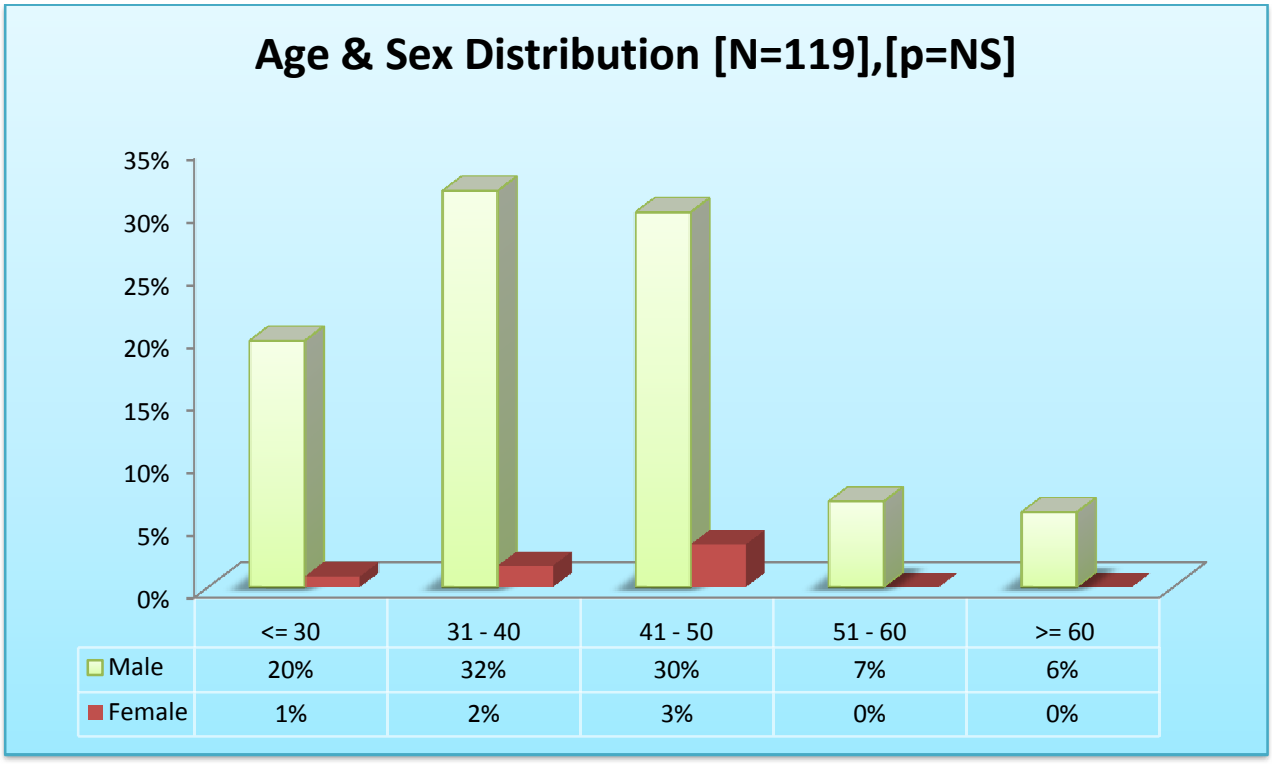
Age	Male	Female	Total
<= 30	23	1	24
31 - 40	37	2	39
41 - 50	35	4	39
51 - 60	8	0	8
>= 60	7	0	7
Total	110	7	117

The above tabular column 1 gives the age sex distribution of the disease in the various age group.

Table 2 – Mean SD for Age

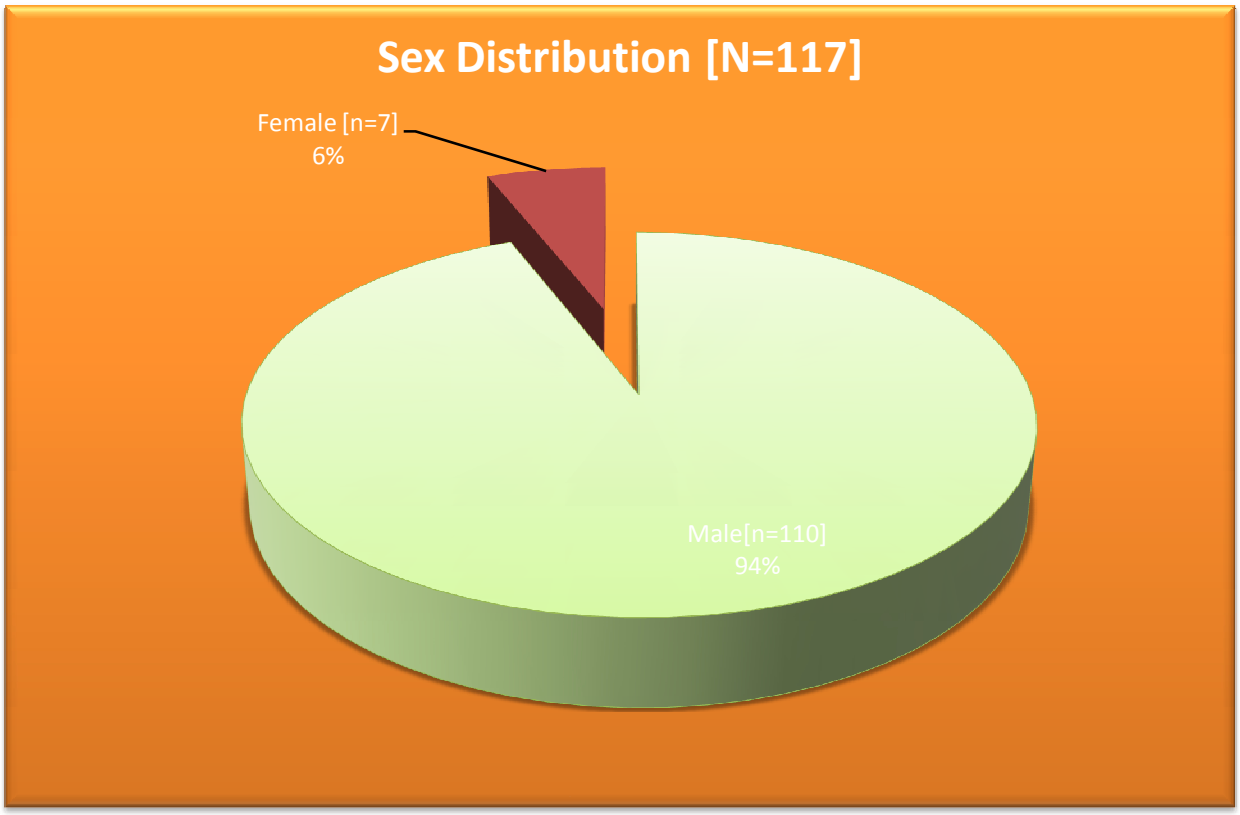
Sex	N	Mean +/- SD	Range
Male	110	39 +/- 12	16 - 72
Female	7	39 +/- 9	23 - 50
Total	117	39 +/- 11	16 - 72

The above tabular column gives the mean and the SD for the age. The explanation is as follows.



The total patients studied in this study were 117, which comprises of 110 males and 7 females. Among the male population the maximum age group is 31-40. Next comes the 41-50 which includes 35 patients. Among the female population the maximum age group is 41-50 which includes about 4 patients.

The next tabular column 2 clearly shows the mean and the standard deviation of the age group. On the whole it is 39+/-11. It is 39+/- 12 for male and 39+/-9 for female. The bar diagram shows the same in the percentage. Males greater than 60 years of age affected by the disease are 6%. This is the least age group affected.



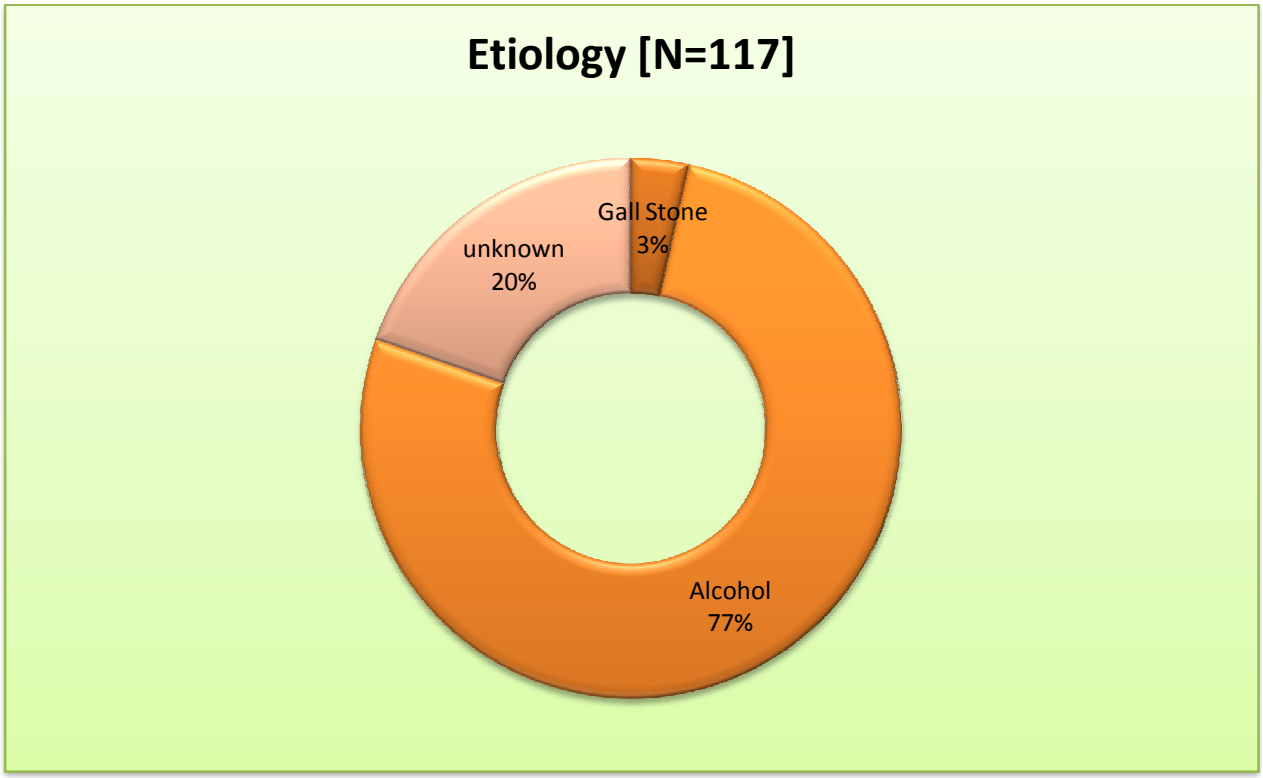
Sex distribution is shown in the pie chart. 6 percent is female and the rest is males. In our population males are commonly affected than the female population. This factor has link with the etiology, in our population alcohol is the common cause of the acute pancreatitis. Alcohol consumption is not prevalent in female population so female are not commonly affected by acute pancreatitis.

B. ETIOLOGY

Table 3 - Etiology

Etiology	No.of cases
Gall Stone	4
Alcohol	90
unknown	23

The tabular column 3 shows the different causes of the acute pancreatitis in prevalent in our population. In our area the most common cause is alcohol. Next it is due to some unknown causes. The unknown cause may be drugs, increase in cholesterol and hypercalcemia. These unknown causes need further analysis, even in unknown causes some patients had history of consumption of alcohol during the earlier days and had stopped consumption of alcohol in recent years. Next common cause is the gallstone. Number of patients who had gall stone pancreatitis is four. On linking with the etiology with the sex incidence we could find out that the gall stone pancreatitis is more common in the females.



The above diagram shows the etiology of the acute pancreatitis in the percentage. The orange shaded area shows that the alcohol contributes the 77% of the etiology in acute pancreatitis. The brown shaded area gives the percentage of gall stone contributing to pancreatitis, it is about 3%. Out of 7 females 2 had gall stones pancreatitis. The gall stone pancreatitis is common in females. Unknown causes contribute to about 20%.

C.SEVERE ACUTE PANCREATITIS(SAP) Vs DIFFEENT PROGNOSTIC SCORE

Table 4 - Severe Acute Pan. vs Prognostic Score

RANSON	SAP		Total	P VALUE
	Yes	No		
< 3	14	67	81	NS
>= 3	11	25	36	
Total	**25**	**92**	**117**	
BISAP				
< 3	10	87	97	NS
>= 3	15	5	20	
Total	**25**	**92**	**117**	
CTSI				
< 3	17	61	78	NS
>= 3	8	31	39	
Total	**25**	**92**	**117**	

The above tabular column 6 shows the distribution of the SAP within and above the cut off value of the different prognostic score.

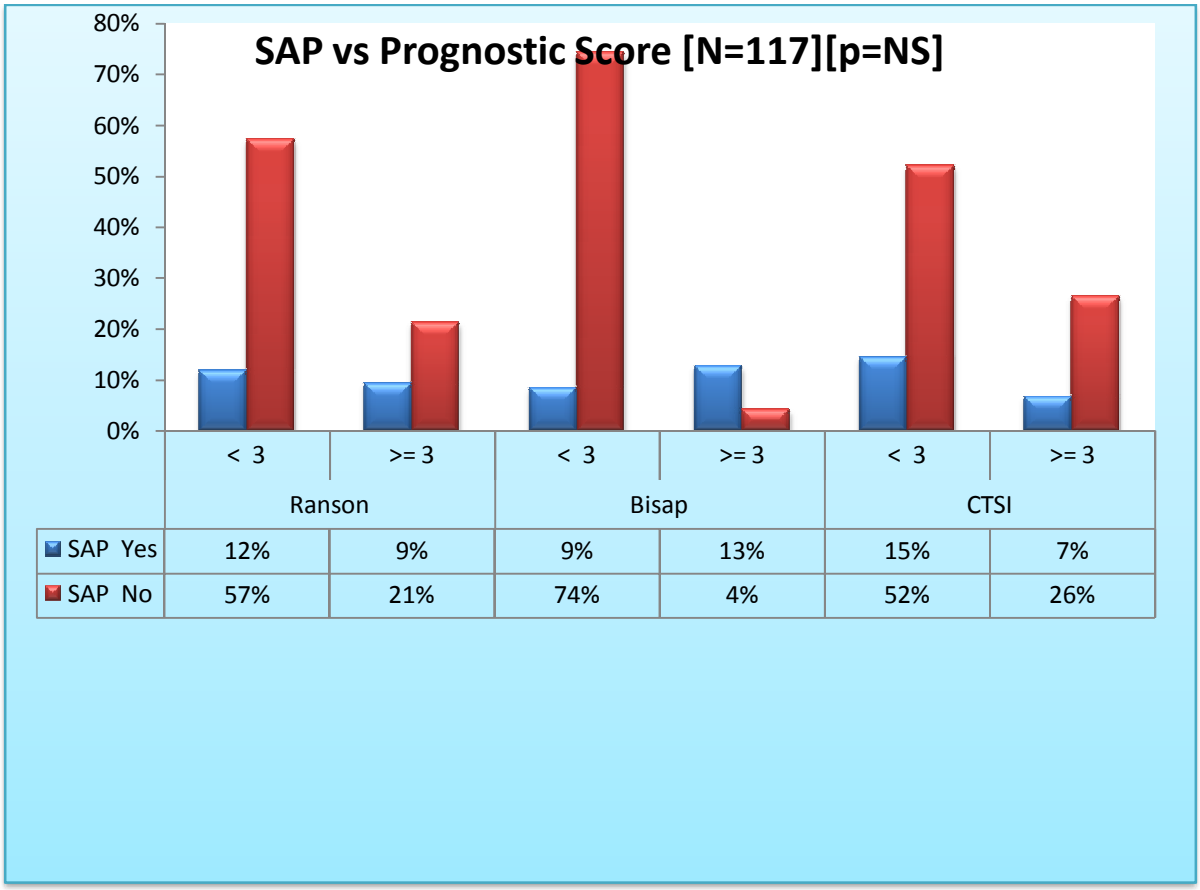
Table 5 - Severe Acute Pain vs Prognostic Score

Study	SEN	SPEC	PPV	NPV	RR	ODDS
RANSON	44%	73%	31%	83%	1.768	2.106
BISAP	60%	95%	75%	90%	7.275	26.100
CTSI	32%	66%	21%	78%	0.941	0.926

TABLE 4 shows the different scores of patients with SAP. 14 patients with SAP had RANSONs score < 3 and 11 with SAP had RANSON score > 3. But only 10 patients with SAP had BISAP score < 3 and 15 SAP patients had BISAP score > 3. CTSI score in 17 SAP was < 3 and 8 patient with CTSI had > 3.

So out of 25 SAP patient BISAP picked out 15 and RANSON picked 14 and CTSI picked only 8.

On looking the sensitivity specificity, PPV, NPV, and the odds ratio BISAP score has the sensitivity of 60% and the CTSI has the least sensitivity of 32%. The specificity of the BISAP score is 95%.PPV and NPV of the BISAP score is 75%, 90% respectively. The odds ratio is 26. Greater than 1 is said to significant. So BISAP score predicts the SAP well comparing the other score.



The above bar diagram shows the relationship between the severe acute pancreatitis and the other prognostic score. The blue shaded area indicates the presence of the SAP and the red shaded area indicates the absence of the SAP. Among the RANSON score <3, SAP is present in 12% and 57% had no SAP. On the other hand in the patients with BISAP score <3, 9% had SAP and 74% had no SAP. In the patients with BISAP score >=3, 15% had SAP and 4% had no SAP.

D) MORTALITY Vs THE PROGNOSTIC SCORE

Table 6 - Mortality vs Prognostic Score

RANSON	Mortality		Total	P VALUE
	Yes	No		
< 3	3	78	81	
>= 3	4	32	36	
Total	**7**	**110**	**117**	NS
BISAP				
< 3	2	95	97	
>= 3	5	15	20	<0.01
Total	**7**	**110**	**117**	
CTSI				
< 3	4	74	78	
>= 3	3	36	39	NS
Total	**7**	**110**	**117**	

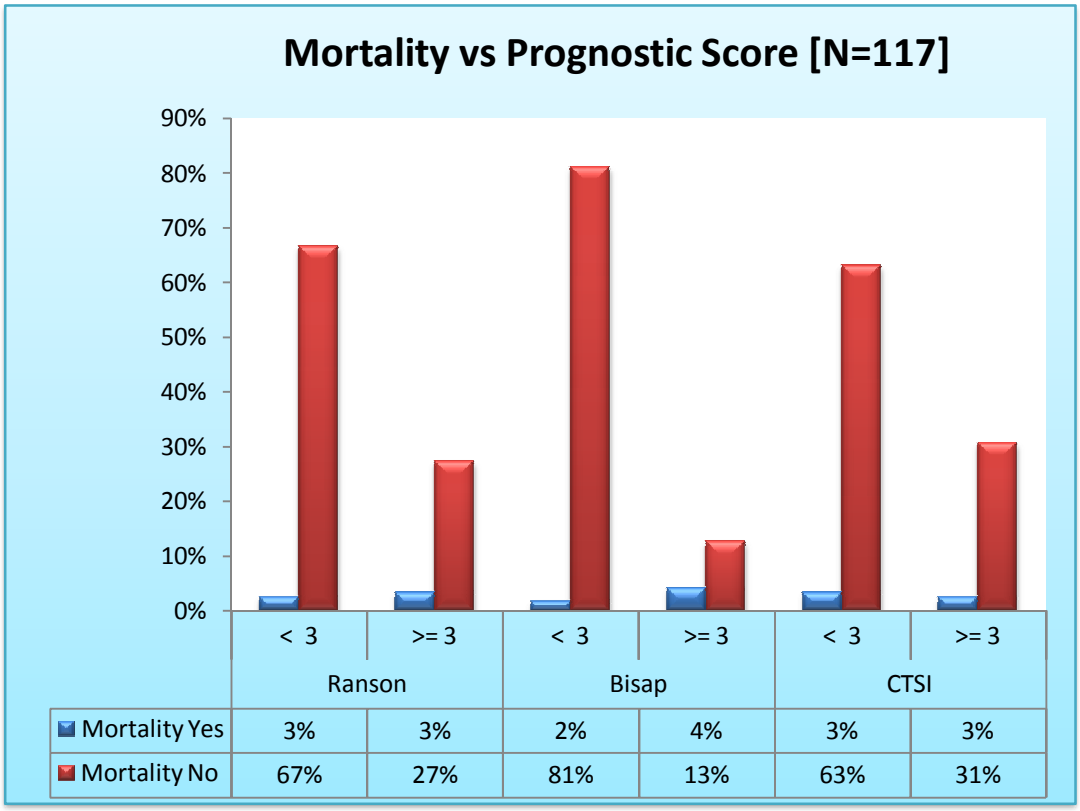
The above tabular column 6 shows the distribution of the mortality within and above the cut off value of the different prognostic score.

Table 7 - Mortality vs Prognostic Score

Study	SEN	SPEC	PPV	NPV	RR	ODDS
RANSON	57%	71%	11%	96%	3.000	3.250
BISAP	71%	86%	25%	98%	12.125	15.833
CTSI	43%	67%	8%	95%	1.500	1.542

This type of analysis shows which type of score is better in predicting the mortality in acute pancreatitis patient. Out of total 7 deaths 4 patients has RANSON score >=3 , 5 patients had BISAP score >=3 and 3 patients had CTSI score >=3. The p value of the BISAP score is <0.01 which shows the significant relationship between the BISAP score and the mortality.

Table 7 shows the sensitivity of the scores in predicting the mortality. It is highest for the BISAP score and it is 71% , the specificity of the score is 86% which is also the highest. While for the RANSON, BISAP score the sensitivity is 57% and 43%, the specificity is 71% and 67% respectively. The PPV and NPV for the BISAP score is 25% and 98% respectively. This shows that BISAP score predicts the mortality well when compared to the other score.



The above bar diagram shows the relationship between the mortality and the various prognostic score. The mortality is three percent for the patients with RANSON score <3 and >=3. There is same percentage of mortality occurred in the patients with CTSI score <3 and >=3. But the mortality is 4% for the patient with BISAP score >=3. This is high when compared to other score. This indicates predicting accuracy of mortality by the BISAP score.

E) PANCRETIC NECROSIS (PANCREATIC NECROSIS) Vs PROGNOSTIC SCORE

Table 8 - Pancreatic Necrosis vs Prognostic Score

RANSON	P Nec		Total	P VALUE
	Present	Absent		
< 3	8	73	81	NS
>= 3	8	28	36	
Total	**16**	**101**	**117**	
BISAP				
< 3	9	88	97	< 0.05
>= 3	7	13	20	
Total	**16**	**101**	**117**	
CTSI				
< 3	2	76	78	<0.001
>= 3	14	26	39	
Total	**16**	**102**	**117**	

The above tabular column 6 shows the distribution of the Pancreatic necrosis within and above the cut off value of the different

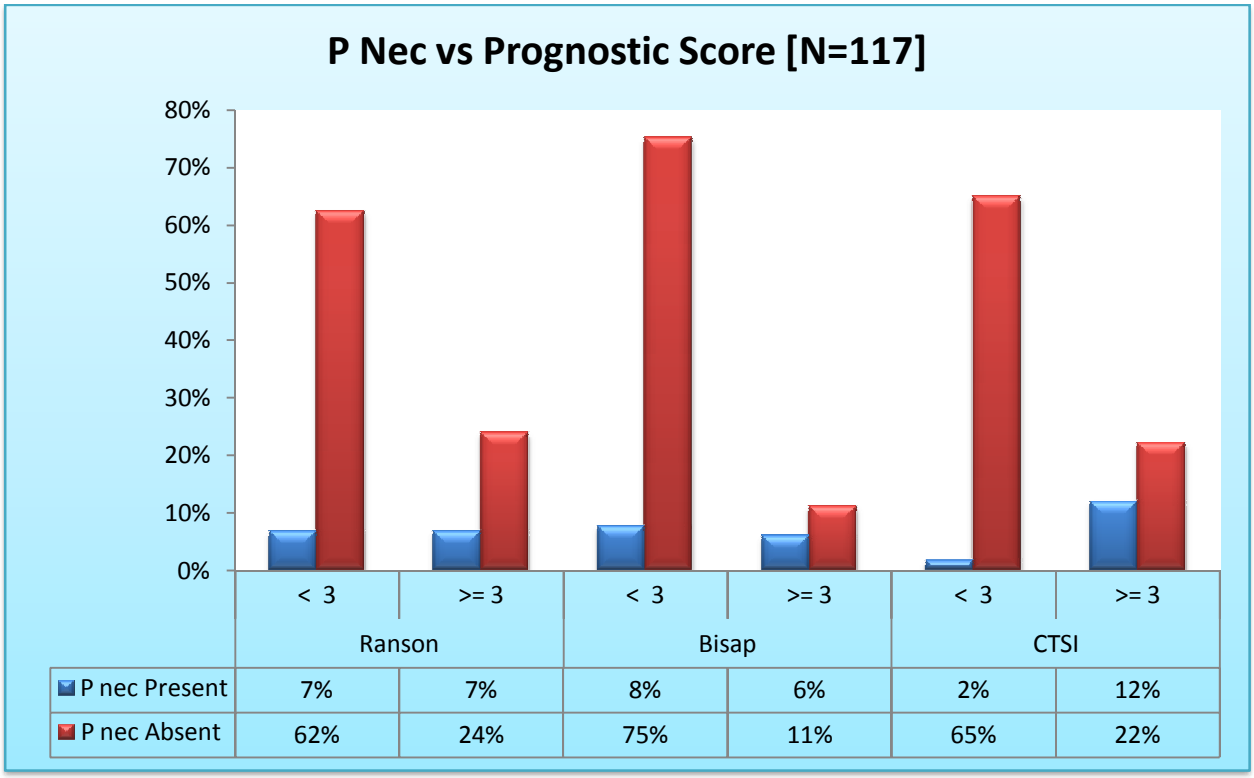
prognostic score.

Table 9 - Pancreatic Necrosis vs Prognostic Score

Study	SEN	SPEC	PPV	NPV	RR	ODDS
RANSON	50%	72%	22%	90%	2.250	2.607
BISAP	44%	87%	35%	91%	3.772	5.265
CTSI	88%	75%	35%	97%	13.650	20.462

TABLE 8 shows the score which can predict the Pancreatic necrosis. Since pancreatic necrosis is a CT finding naturally it shows that CTSI is good in predicting the pancreatic necrosis. Out of the 117 patients 16 patients had pancreatic necrosis. Out of the 16 patients 14 patients had CTSI score >=3 and only 2 patients had score <3. The P value of the BISAP score in predicting the mortality is <0.05. But the P value of the CTSI score in predicting the pancreatic necrosis is <0.001. This indicates strong relationship between the CTSI score and pancreatic necrosis.

The sensitivity and the specificity of the CTSI score in predicting the pancreatic necrosis is 88% and 75% respectively. The odds ratio is 20 which show the strongest relationship between the CTSI and the pancreatic necrosis.



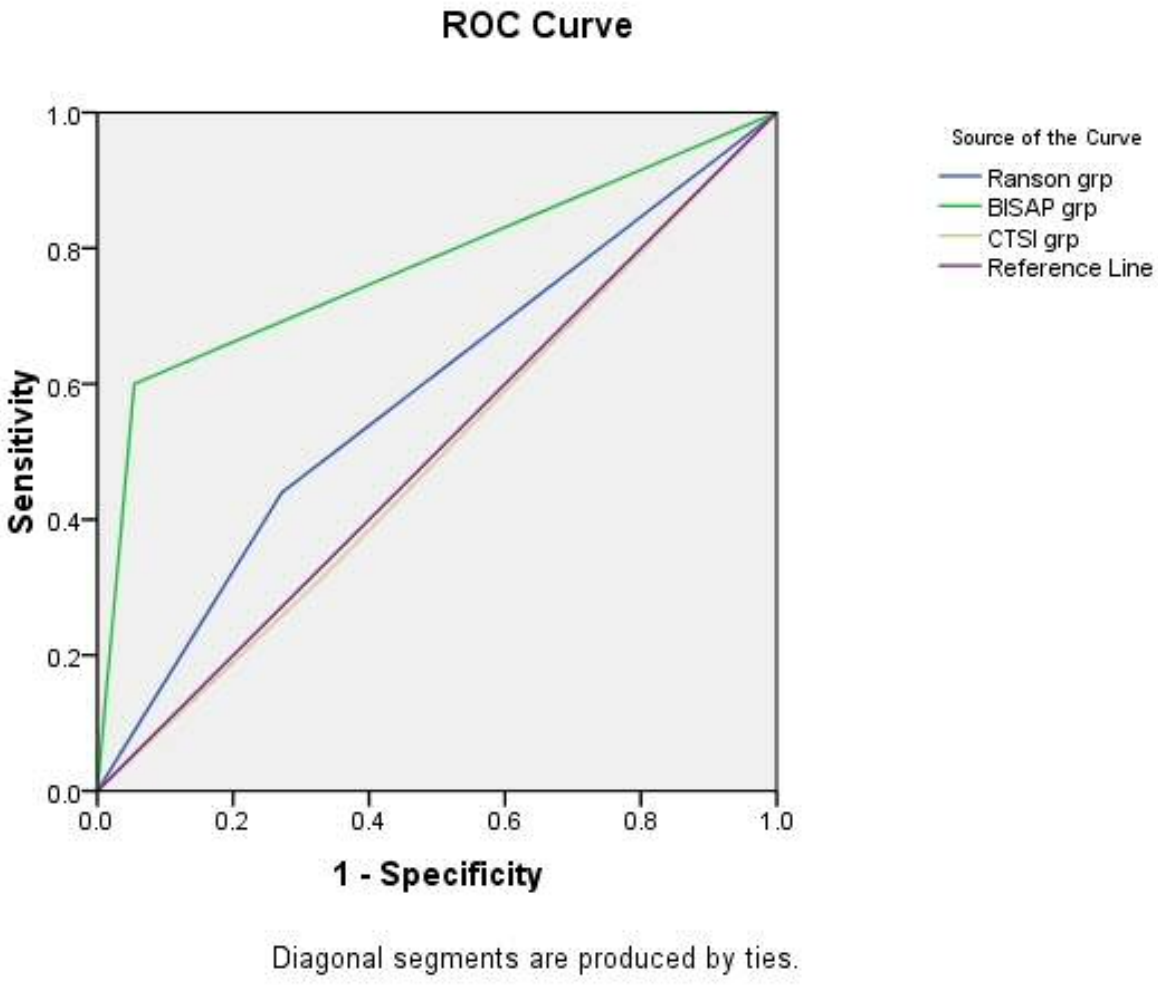
The above bar diagram shows that the 12% of the patients with CTSI >=3 had Pancreatic necrosis. Only 2% of the patients with score < 3 had Pancreatic necrosis. 7% of the patients with RANSON score <3 and >=3 had pancreatic necrosis. 8% of the patients with BISAP score < 3 had pancreatic necrosis. Six percent of the patients with score >=3 had pancreatic necrosis. The bar diagram finally shows the highest predicting value for pancreatic necrosis by the CTSI.

F)MORBIDITY

Morbidity is assessed by number of days patients stayed in the hospital. The mean and standard deviation is 9.03+/- 3.42 days. The overall days patients stayed in the hospital has no significant relationship with the scoring system.

AREA UNDER CURVES

AUC 1 COMPARISON OF SCORING SYSTEMS IN PREDICATING SEVERE ACUTE PANCREATITIS



AUC FOR SAP

The above graph is the ROC for SAP. The green colored line shows the BISAP score, the blue colored line shows the RANSON score, the pink colored indicates the CTSI and the violet colored line is the reference line. The BISAP score line is high above all line, the area

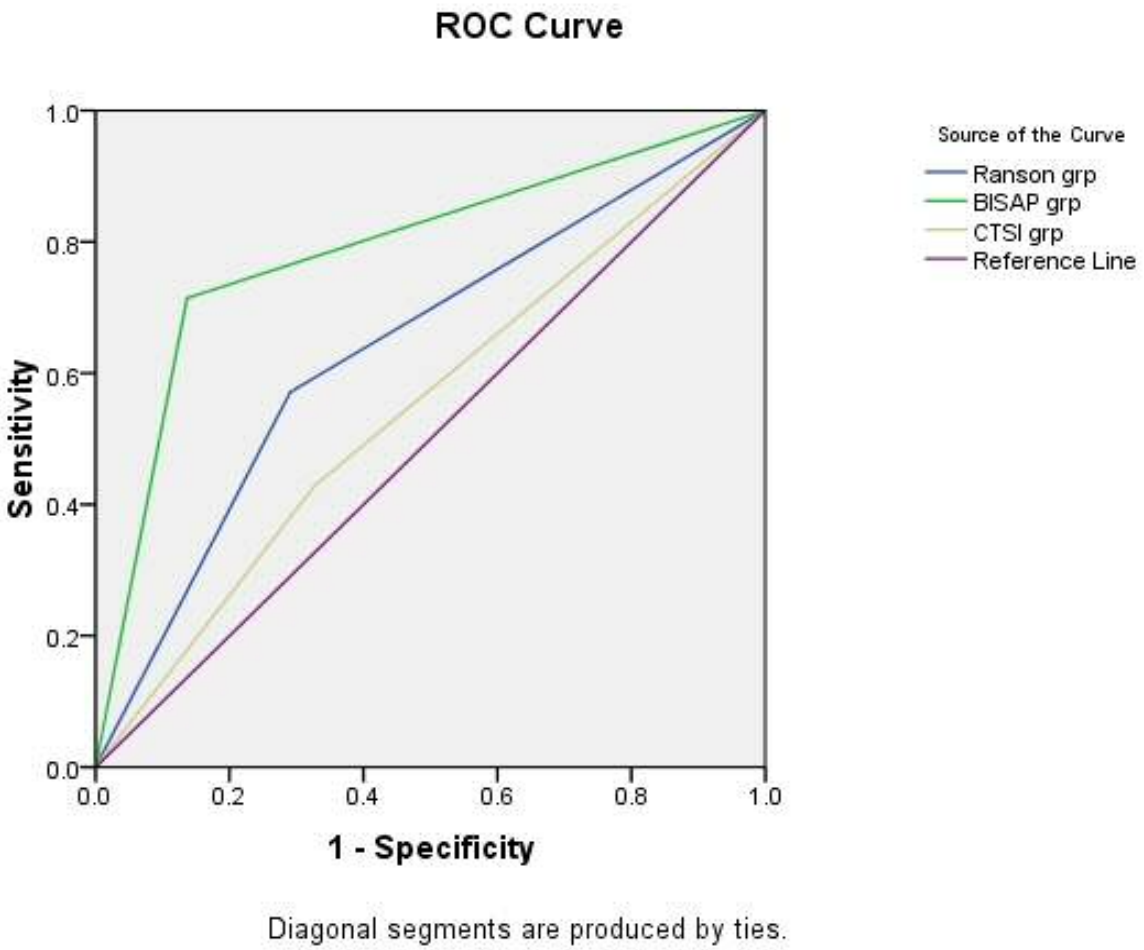
covered below the line is maximum when compared with other line Source of the graph is RANSON, BISAP, CTSI group. The AUC is 0.773 for BISAP which is the maximum when compared to other scores (table 14). The standard error of the same score is 0.0653 and the 95% confidence interval is 0.650 to 0.896. The AUC is least for CTSI group in predicting the SAP.

Table 10 - Area Under the Curve for SAP

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
RANSON grp	0.584	0.066	0.198	0.454	0.714
BISAP grp	0.773	0.063	0.000	0.650	0.896
CTSI grp	0.492	0.065	0.897	0.364	0.619

The Table 10 shows that BISAP score is good in predicting the SAP in the acute pancreatitis patient.

AUC 2 COMPARISION OF THE SCORING SYSTEM IN PREDICTING THE MORTALITY



The ROC curve 2 finds out the score which predicts the mortality in better way. The above graph is the ROC for mortality. The source of the graphs is RANSON, BISAP, CTSI group. The green colored line shows the BISAP score, the blue colored line shows the RANSON score, the pink colored indicates the CTSI and the violet colored line is

the reference line. The BISAP score line is high above all line, the area covered below the line is maximum when compared with other line.

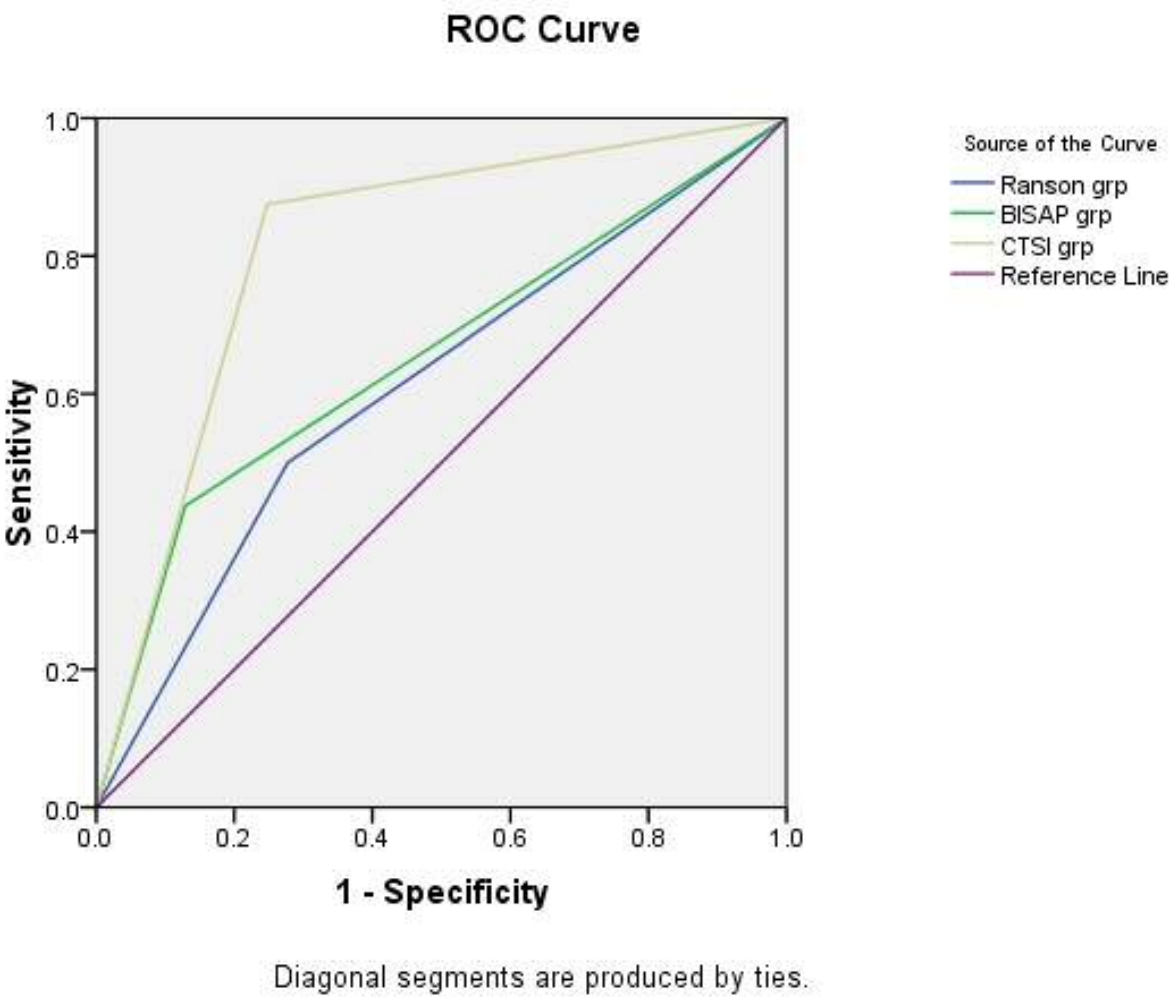
Table 11 - Area Under the Curve for mortality

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
RANSON grp	0.640	0.112	0.215	0.420	0.860
BISAP grp	0.789	0.102	0.011	0.589	0.989
CTSI grp	0.551	0.115	0.654	0.326	0.776

The AUC for the BISAP is 0.789 and it has the standard error of 0.102. the AUC for the RANSON and the CTSI is 0.640 and 0.551 respectively. The BISAP score has the CI of 0.589 to 0.989.

So this clearly suggests that BISAP score is better when compared to the other score.

AUC 3 COMPARING THE SCORING SYSTEM IN PREDICTING THE PANCREATIC NECROSIS



The ROC curve 2 finds out the score which predicts the pancreatic necrosis. The above graph is the ROC for pancreatic necrosis. The source of the graph is RANSON, BISAP, CTSI group. The green colored line shows the BISAP score, the blue colored line

shows the RANSON score, the pink colored indicates the CTSI and the violet colored line is the reference line. The CTSI score line is high above all line, the area covered below the line is maximum when compared with other line

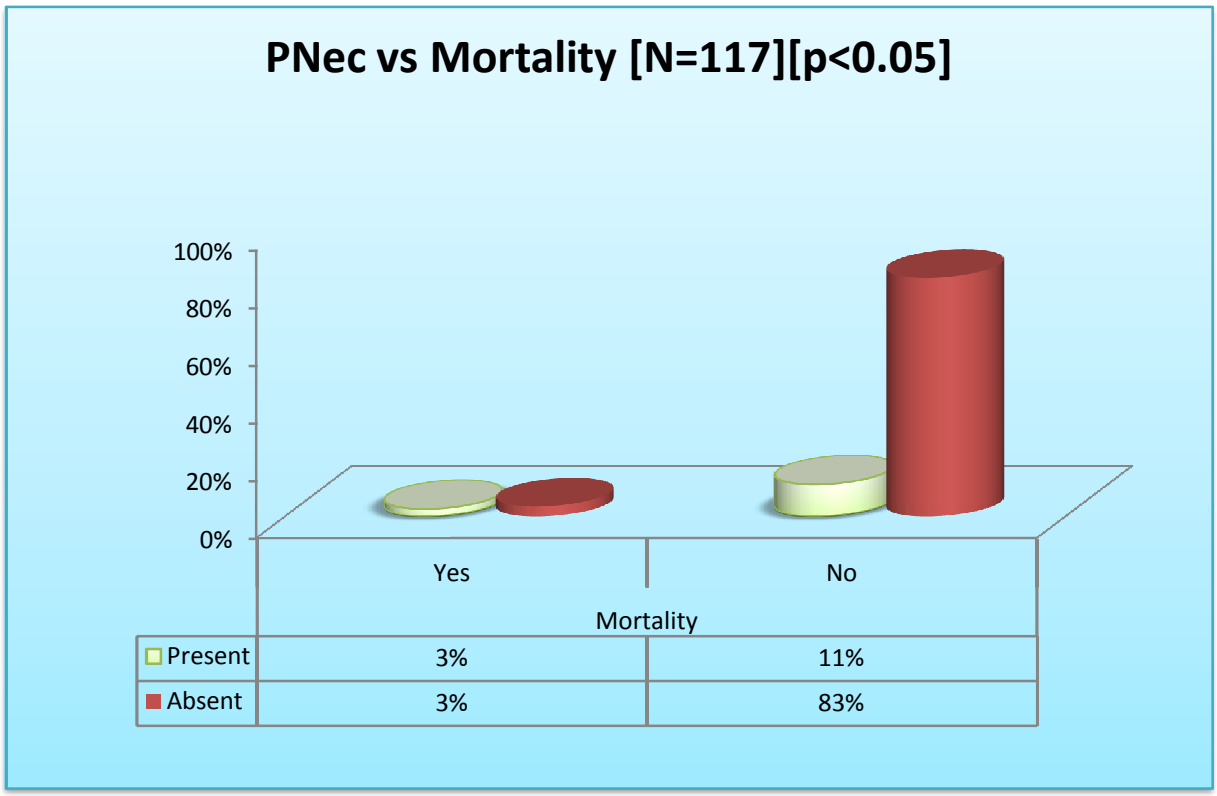
Table 11 - Area Under the Curve for pancreatic necrosis

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
RANSON grp	0.611	0.079	0.153	0.457	0.766
BISAP grp	0.654	0.082	0.048	0.494	0.815
CTSI grp	0.814	0.055	0.000	0.706	0.921

The ROC graph 3 finds which score predicts the pancreatic necrosis. The AUC for CTSI group is 0.814 and the 95% CI for the same group is 0.706 and 0.921. AUC is least for the RANSON group and it is 0.611. BISAP group has the AUC of 0.654 and the standard error is 0.082.This shows that CTSI predicts the pancreatic necrosis well.

ATLANDA CLASSIFICATION ANALYSIS

The Atlanta classification classifies the acute pancreatitis into necrotic pancreatitis and edematous pancreatitis. The classification uses the above parameter to predict the mortality. In this study of 117 patients necrotic pancreatitis is 16 i.e. 14% and the rest is edematous pancreatitis accounting to about 86%.



The above bar diagram compares the mortality and the pancreatitis necrosis and the mortality. Among 16 patients who had pancreatic necrosis 3 died and the others survived. Among the rest 4 died and 83% survived. Only 11% of the patients who had acute severe pancreatitis survived (bar diagram).

This shows there relationship between the pancreatic necrosis and the mortality.

PANCREATIC NECROSIS vs SEVERE ACUTE PANCREATITIS

Table 12 - PANCREATIC NECROSIS VS SAP

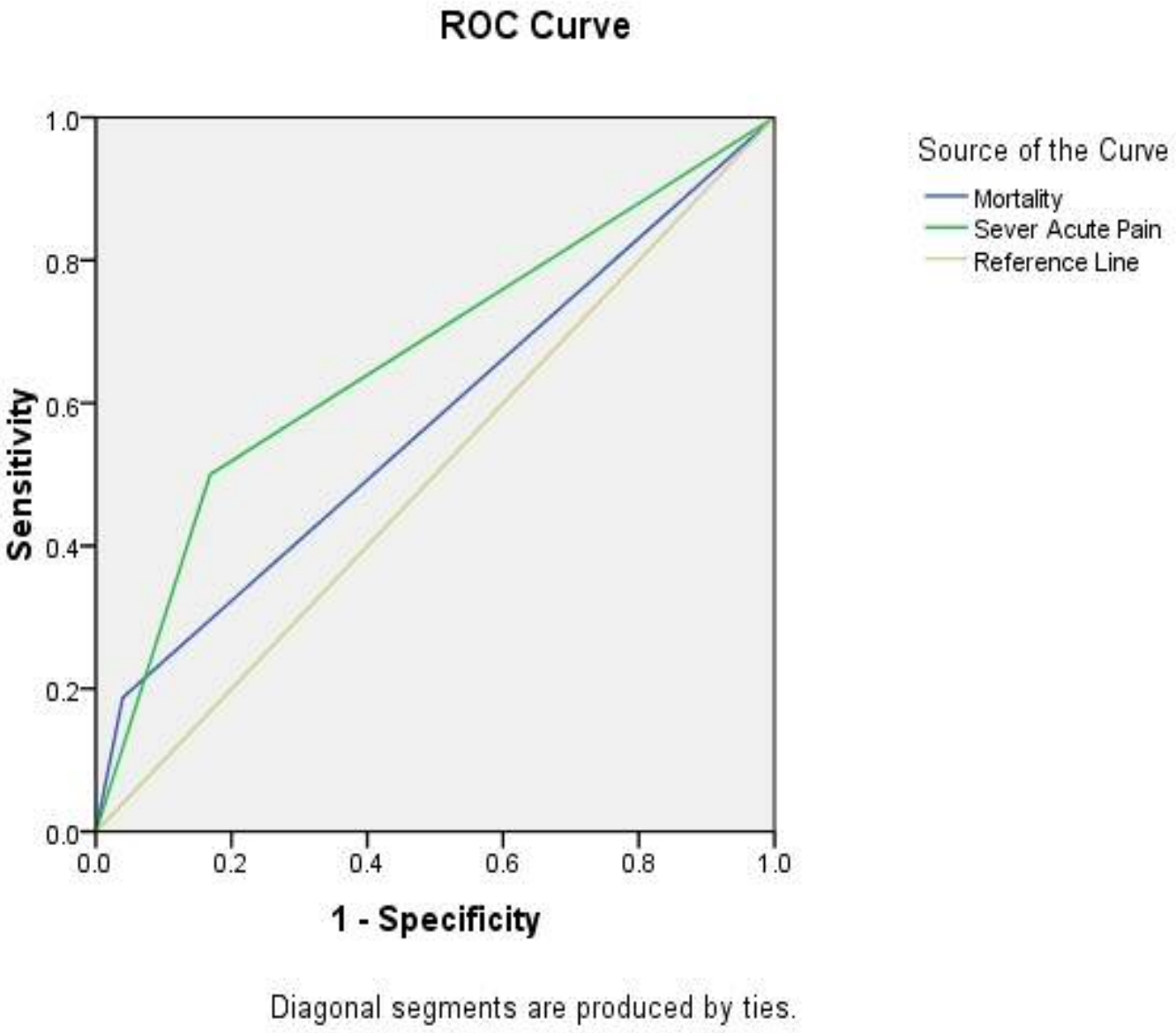
P Nec	SAP		Total	(%)
	Yes	No		
Present	8	8	16	14%
Absent	17	84	101	86%
Total	25	92	117	100%

This tabular column 12 gives the analysis between the pancreatic necrosis and the SAP. Out of the 25 SAP patients pancreatic necrosis present in 8 patients and absent in 17 patients. On the other hand out of 92 patients with no SAP, 8 patients had pancreatic necrosis.

PAN NEC VS SAP & MORTALITYAUC

This graph discusses the factor pancreatic necrosis in predicting the mortality and the severity since the pancreatic necrosis is the only factor used in

PAN NEC VS SAP & MORTALITYAUC



the ATLANDA classification. The green colored line indicates the SAP, the violet line indicated the mortality and the other line is the mortality. The AUC for the factor mortality within the pancreatic necrosis is 0.574, which is much less when compared to the AUC for the severity. This suggests that the patients with the pancreatic necrosis the severe pancreatitis is likely.

DISCUSSION

1 AGE COMPARISON

Table 13 - Age Comparison

S.No	STUDY	MEAN AGE
1	PRESENT STUDY	39+/-11
2	VIKESH SINGH AND GROUP[13]	52+/-16
3	GEORGIOS AND GROUP	52+/-14

The mean age with SD of study group is 39+/-11 and inter quartile range is 16-72. This is less when compared to other. This might be due to the etiology alcohol is more prevalent in the younger age group.

2. ETIOLOGY

In our study the most common etiology is alcohol but in other study the most common etiology is gall stones. In our study gall stone is most common etiology in females.

2. MEDIAN HOSPITAL STAY

Table 14 – Median Hospitals

S.No	STUDY	HOSPITAL STAY(days)
1	PRESENT	9
2	A.O FARRELL	7

Compare to the study done by AO Farrell[25] the hospital stay is increased with our study.

4. MORTALITY COMPARITION

Table 15 – Mortality Comparison

S.No	STUDY	MORTALITY%
1	PRESENT STUDY	5.9
2	VIKESH SINGH AND GROUP	3.5
3	GEORGIOS AND GROUP	3.5
4	LOSADA M AND GROUP[12]	9
5	SHEN AND GROUP[11]	2.7

Table 16 - SENSITIVITY, SPECIFICITY,PPV,NPV OF BISAP SCORE IN PREDICTING THE MORTALITY IN DIFF. STUDIES

S.NO	STUDY	SENSITIVITY %	SPECIFICITY %	PPV%	NPV%
1	PRESENT STUDY	71	86	25	98
2	VIKESH SINGH AND GROUP	71	83	17.5	99
3	GEORGIOS AND GROUP[15]	57.1	87.6	15.5	98

Table 17 - AUC OF BISAP SCORE IN PREDICTING THE MORTALITY IN DIFF STUDIES

S.No	STUDY	AUC
1	PRESENT STUDY	0.78
2	VIKESH SINGH AND GROUP	0.83
3	GEORGIOS AND GROUP	0.82

The overall mortality of the study is 5.9 it is higher when compared to the Vikesh Singh and group, Georgios and group but lower than the LOSADA M and group. This might be due to the many factors such as availability of the intensive care in the developing country, other co morbid factor. The sensitivity, specificity, PPV, NPV of the BISAP score in predicting the mortality is consistent with study done by Vikesh Singh and group. The sensitivity is higher when compared to the GEORGIOS and group study. This suggests that BISAP score does well in predicting the mortality. AUC for predicting the mortality by the BISAP score is also more or less consistent with the other study.

4. SAP COMPARISON IN DIFFERENT STUDIES

Table 18 - SENSITIVITY, SPECIFICITY, PPV, NPV OF BISAP SCORE IN PREDICTING THE SAP

S.NO	STUDY	SENSITIVITY	SPECIFICITY	PPV	NPV
1	PRESENT STUDY	60	95	75	90
2	GEORGIOS AND GROUP[15]	35.5	92.4	57.7	84.3

The above tabular column 19 compares the sensitivity, specificity, PPV, NPV

of the BISAP score in predicting the SAP with other study. The sensitivity and specificity in the present study is 60% and 95% respectively. While in the georgious group the same is 35-5% and 92.4% respectively. The specificity is consistent when compared with present study.

Table – 19 AUC OF BISAP SCORE IN PREDICTING THE SAP IN DIFF STUDIES

S.NO	STUDY	AUC
1	PRESENT STUDY	0.773
2	GEORGIOS AND GROUP	0.8I

In our study BISAP score performed well compared to the Georgiou and group study. However the AUC of the later study is more when compared with our study. SAP also well predicted by BISAP in our study. This indicates BISAP score is able to predict development of the organ failure in first 24 hour. In The study performed by Vikesh shingh it was found that patients with > = 3 score in BISAP had 7.4 times more prone to develop SAP.

Table 20 - SENSITIVITY, SPECIFICITY, PPV, NPV OF BISAP SCORE IN PREDICTING THE PANCREATIC NECROSIS IN DIFF STUDIES

S. NO	SCORE	STUDY	SENTI	SPECI	PPV	NPV
1	BISAP	PRESENT STUDY	50	72	22	90
		GEORGIOS AND GROUP	33.3	90.6	46.6	84.9
2	CTSI	PRESENT STUDY	88	75	35	97
		GEORGIOS AND GROUP	97.2	75.8	59.3	98.7

Pancreatic necrosis is well predicted by the CTSI in both the study. But BISAP score has increased sensitivity in predicting the pancreatic necrosis when compared to the GEORGIOS and group.

5. ATLANDA CLASSIFICATION ANALYSIS

Table 21 – Atlanta Classification analysis

S.No	STUDY	NECROTIC PAN	OTHERS
1	PRESENT STUDY	14	86
2	LOSADA M STUDY[12]	32	68

According to Atlanta classification 80% of the pancreatitis belongs to acute edematous pancreatitis i.e. the mild form. Rest of the pancreatitis belongs to the necrotizing pancreatitis. Our study is consistent with the Atlanta classification. Further the Atlanta classification states that mortality in the necrotizing type is 15%-30%, in our study out of 16 patients who had the necrotizing 3 died. This accounts to about 18.75% which is also within the limits of the classification.

CONCLUSION

To conclude BISAP score is simple and it is the better scoring system in predicting the prognosis when compared to other score. The BISAP score has many advantages when compared to other scoring system. The advantages are as follows

1) It is simple in calculating the score the components are clinically relevant and easy to obtain.
2) It uses the 24 hrs data to predict the prognosis of the acute pancreatitis patient. But in RANSON score 48hrs data has to be collected.
3) BISAP SCORE gives weightage to the immune response of the injury and includes the age criteria.
4) BISAP score is used to triage the patient to closer observation and to asses which patients need closer monitoring.

Atlanta classification also holds good for the classify the pancreatitis into mild and severe disease also holds good to assess the prognosis of the acute pancreatitis. On the whole when BISAP scoring system is combined with the ct scan then the assessment of the acute pancreatitis becomes very accurate. When combined they can used triage the patient, anticipate the complications, assess the severity, predict the mortality.

SUMMARY

1. In our study occurrence of the pancreatitis is common in the age group is 39+/-11.
2. Most common etiology is alcohol it is about77%.
3. In our community men are most affected than women.
4. Median hospital stay in our community for acute pancreatitis is about 9+/-3days.
5. According to ATLANDA score 86% had mild disease.
6. On the whole BISAP score is better in predicting the mortality an severe disease .
7. CTSI predicted the necrotic pancreatitis in a better way.
8. Among the pancreatic necrosis the mortality is 3%.
9. Among the pancreatic necrosis patient 7% had severe acute pancreatitis.
10. The sensitivity and specificity of the BISAP score in predicting the severe acute pancreatitis is 60%, 95%.
11. The sensitivity and specificity of the BISAP score in predicting the mortality is 71%, 86%.
12. The sensitivity and specificity of the CTSI score in predicting the pancreatic necrosis is 88%, 75%.

The below tabular column 23 summarizes the ability of the prognostic score in predicting the SAP. On the whole the SAP is 21%. In the patients with score >=3 in RANSON, BISAP ,CTSI scoring system there is 31%,17%,33% SAP respectively.

Table 22 - SEVERE ACUTE PAN VS PROGNOSTIC SCORE

RANSON	**SAP**		**Total**	**P VALUE**
	Yes	**No**		
< 3	12%	57%	69%	NS
>= 3	9%	21%	31%	
Total	**21%**	**79%**	**100%**	
BISAP				
< 3	9%	74%	83%	NS
>= 3	13%	4%	17%	
Total	**21%**	**79%**	**100%**	
CTSI				
< 3	15%	52%	67%	NS
>= 3	7%	26%	33%	
Total	**21%**	**79%**	**100%**	

The below tabular column 24 summarizes the ability of the prognostic score in predicting the mortality. On the whole the mortality is 5.9%. In the patients with score >=3 in RANSON, BISAP ,CTSI scoring system there is 31%,17%,34% mortality respectively.

TABLE 23-MORTALITY VS PROGNOSTIC SCORE

RANSON	Mortality		Total	P VALUE
	Yes	No		
< 3	3	78	81	
>= 3	4	32	36	
Total	**7(5.9%)**	**110**	**117**	NS
BISAP				
< 3	2	95	97	
>= 3	5	15	20	<0.01
Total	**7(5.9%)**	**110**	**117**	
CTSI				
< 3	4	74	78	
>= 3	3	36	39	NS
Total	**7(5.9%)**	**110**	**117**	

The below tabular column 25 summarizes the ability of the prognostic score in predicting the pancreatic necrosis. On the whole the pancreatic necrosis is 14%. In the patients with score >=3 in RANSON, BISAP ,CTSI scoring system there is 31%,17%,34% pancreatic necrosis respectively.

Table 24 - PANCREATIC NECROSIS VS PROGNOSTIC SCORE

RANSON	**P Nec**		**Total**	**P VALUE**
	Present	**Absent**		
< 3	7%	62%	69%	NS
>= 3	7%	24%	31%	
Total	**14%**	**86%**	**100%**	
BISAP				
< 3	8%	75%	83%	< 0.05
>= 3	6%	11%	17%	
Total	**14%**	**86%**	**100%**	
CTSI				
< 3	2%	65%	67%	<0.001
>= 3	12%	22%	34%	
Total	**14%**	**87%**	**101%**	

The below tabular column summarizes the AUC of the various scoring system in predicting the SAP, mortality, pancreatic necrosis.

Table 25 - AREA UNDER CURVE

SAP	**Area**	**95% CI**	**p value**
RANSON	0.584	0.454-0.714	NS
BISAP	0.773	0.650-0.896	<0.001
CTSI	0.492	0.364-0.619	NS
Mortality			
RANSON	0.640	0.420-0.860	NS
BISAP	0.789	0.589 - 0.989	<0.05
CTSI	0.551	0.326 - 0.776	NS
P Nec			
RANSON	0.611	0.457 - 0.766	NS
BISAP	0.654	0.494 - 0.815	<0.05
CTSI	0.814	0.706 - 0.921	<0.001

BIBILIOGRAPHY

1. Susan Standring PhD DSc. The 39th edition of Gray's Anatomy ; 88 pancreas: spleen and suprarenal and spleen
2. Michael J. Zinner, MD, FACS. Maingot's Abdominal Operations 11TH edition. Chapter 36; Management of Acute Pancreatitis
3. Banks PA – acute pancreatitis ; medical & surgical management
4. Beger H G et al- natural course of acute pancreatitis; world j surg;21:130-135
5. Sakorafas GH – ETIOLOGY and pathogenesis of acute pancreatitis; j clin gastro 2000;30: 343-356
6. RANSON J : diagnostic standards of acute pancreatitis; world j surg 1997 ; 21: 136
7. Balthazar EJ, RANSON JH. Acute pancreatitis: value of CT in establishing prognosis. Radiology 1990;174(2):331–6.
8. Balthazar EJ, Freeny PC, van Sonnenberg E. Imaging and intervention in acute pancreatitis. Radiology 1994;193:297–306
9. Golub R, et al. Role of antibiotics in acute pancreatitis: a meta-analysis. J Gastrointest Surg 1998;2(6):496–503
10. Niesel HC, et al. Epidural blockade for analgesia and treatment of acute pancreatitis. Reg Anaesth 1991;14(6):97–100

11. Shen, Hsiu-Nien; Lu, Chin-Li. Incidence, Resource Use, and Outcome of Acute Pancreatitis With/Without Intensive Care: A Nationwide Population-Based Study in Taiwan Source: Pancreas Vol: 40, No: 1, January, 2011 [Page 10-15]
12. Losada M, Jorge. Retrospective Review of 251 Patients with Acute Pancreatitis ; Source: Revista Chilena de Cirugía Vol: 62, No: 6, December, 2010 [Page 557-563]
13. Singh VK, Wu BU, Bollen TL et al. A prospective evaluation of the bedside index for severity in acute pancreatitis score in assessing mortality and intermediate markers of severity in acute pancreatitis. Am J Gastroenterol 2009;104:966–971.
14. Ranson JH, Rifkind KM, Roses DF, et al. Prognostic signs and the role of operative management in acute pancreatitis. Surg Gynecol Obstet 1974;139:69–81
15. Georgios I Papachristou, Venkata Muddana, Dhiraj Yadav .Comparison of BISAP, RANSON's, APACHE-II, and CTSI Scores in Predicting Organ Failure, Complications, and Mortality in Acute Pancreatitis;volume- 105: 435-441.
16. Corsetti J, Cox."Combined serum amylase and lipase determinations for diagnosis of suspected acute pancreatitis". Clin Chem 39 (12): 2495–9.
17. Bockus gastroenterology, chronic pancreatitis, Marks, Simmy Bank et al. 4, vol. 6: 4021.

18. Norman S. Williams MS FRCS FMed Sci . Baily and Loves, Short practice of surgery, 25th edition;64 THE PANCREAS.
19. Mastery of surgery, liyod m.nyhus m.d., robert j.baker m.d., charles f. frey, partial and subtotal pancreatectomy for pancreatitis, vol.2 : 780
20. Textbook of surgery, Davis and Christopher, Pancreatitis by Edward L.Bradley III, M.D., et al. 12th edition vol.1 :1300
21. Brooks J.R. and Gifford G.H. Pancreatic Homotrons Plantation, TRANSPLANT BUL, vol.6: 103, 1959.
22. Sutherland D.E., Pancreatic islet cell transplatation, Surg. Cli. of North America, 58 : 365, 1978.
23. Whipple A.O. Radical surgery for certain cases of pancreatic fibrosis associated with calcerous deposits, Ann. Surg. 124: 991, 1946.
24. Roger et al., Treatment of Chronic alcoholic pancreatitis by Pancreatic resection., Amm J. Surg., 156 : 162, 1959. 31. White et al., Treatment of pancreatitis by left Splaninectomy and Coeliac ganglinectomy, Amm J. Surg., 112: 195, 1966.
25. Forsmark CE, Baillie J. AGA Institute technical review on acute pancreatitis. Gastroenterology 2007;132:2022–2044.

PERFORMA FOR ACUTE PANCREATITIS

NAME; AGE: SEX: PHONE NO:

D.O.A:

ADDRESS:

COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

HISTORY OF PAST ILLNESS:

GENRAL EXAMINATION:

ABDOMEN EXAMINATION:

- Inspection
- Palpation
- Percussion
- Auscultation

SERUM AMLYASE:

1. RANSON SCORE

ADMISSION	48 HRS
1 AGE	1. HB DECLINE:
2 W.B.C:	2. BUN INCREASE
3 SUGAR	3. FLUID REPLACED
4AST:	4. PAO2
5LDH:	5. SERUM CALCIUM

2. BISAP SCORING

BUN: GCS: AGE: PLEURAL EFFUSION:

SIRS

TEMP: RR: PR: WBC:

3. BALTHAZAR SCORE:

1 NORMAL CT

2 DIFFUSE ENLARGEMENT OF PANCREAS

3 PANCREATIC GLAND ABNORMALITY &INFLATION

4 FLUID COLLECTIONS IN SINGLE LOCATION

5 > 2 FLUID COLLECTION

% OF PANCREATIC NECROSIS

4. ATLANDA CLASSIFICATION;

1 ACUTE EDEMATOUS PANCREAS;

2 ACUTE NECROTIZING PANCREAS

1 RANSON SCORE ETIOLOGY

3 BISAP SCORING

5 BALTHAZAR SCORE:

SAP

PAN NEC

MORTALITY MORBIDITY

BISAP SCORE VALUES

Sl.no.	Name	Age	BuN Mg/dl	GCS Fr 15	AGE	PL.EF	SIRS	BISAP	SAP	Mortal	Panc Nec
1	VIJAY	37	28	15	0	0	1	2	0	0	0
2	MATHEWS	63	30	15	1	1	1	4	1	1	1
3	SIVA	16	21	15	0	0	1	1	0	0	0
4	KULANTHAISAMY	43	22	15	0	0	0	0	0	0	0
5	GOWRISANKER	23	30	10	0	0	1	3	1	1	0
6	RAMESHKUMAR	25	29	15	0	0	1	2	0	0	0
7	HUSSAIN	38	28	15	0	0	1	2	0	0	0
8	CHINNAN	53	27	15	0	0	0	1	0	0	0
9	KARUPPUSAMY	48	30	15	0	0	1	2	0	0	0
10	PRAKASH	32	31	10	0	0	1	3	1	0	0
11	KARUPPUSAMY	44	30	15	0	0	1	2	0	0	0
12	KARTHIKAYEN	65	29	15	1	0	0	2	0	0	0
13	MURUGESAN	23	32	10	0	1	1	4	1	0	1
14	ARUMUGAM	62	25	15	1	0	0	1	0	0	0
15	SENTHIL KUMAR	65	24	15	1	1	1	5	0	0	0
16	KRISHNAN	25	18	15	0	0	0	0	0	0	0
17	MUTHUKUMARSAY	48	28	15	0	0	1	2	0	0	0
18	JOHN BOSCO	20	25	15	0	0	1	2	0	0	0
19	SENTHIL KUMAR	22	34	15	0	0	0	1	1	0	1
20	SIDDIQUE	28	31	15	0	0	1	2	0	0	0
21	MOHAMAD	39	23	15	0	0	0	1	0	0	0
22	GOWRISANKER	37	28	15	0	0	0	1	0	0	1
23	RAMESH	33	30	10	0	0	0	2	0	0	0
24	BAKER	44	22	15	0	0	0	0	0	0	0
25	GANESH	26	33	10	0	0	1	3	0	0	0
26	SURESH	42	28	15	0	0	1	2	1	0	1
27	ALAGAPPAN	45	27	15	0	0	1	2	0	0	0
28	RAMALINGAM	39	28	10	0	0	0	2	0	0	0
29	SENTHIL VEL	22	29	15	0	0	0	1	0	0	0
30	BANNARI	34	33	15	0	1	1	3	1	0	0
31	KANNAN	44	30	15	0	0	0	1	0	0	0
32	THANGARAJ	27	21	15	0	0	0	0	0	0	0
33	DURAIRAJ	32	29	15	0	0	1	2	0	0	0
34	ARUMUGAM	23	28	15	0	0	1	2	0	0	0
35	KARRUPUSAMY	52	33	15	0	0	0	1	0	0	0
36	VELUSAMY	38	34	5	0	1	1	4	1	1	0
37	CHANDRAKALA	23	26	15	0	0	1	2	0	0	1

38	RAKIYYAPAN	37	27	15	0	0	0	1	0	0	0
39	SARAGANAN	39	29	15	0	0	1	2	0	0	0
40	UHAITHULA	39	21	15	0	0	0	0	0	0	0
41	VELAVANTHAN	50	26	15	0	0	0	2	0	0	0
42	SUBRAMANI	45	33	5	0	1	1	4	0	0	1
43	AROKIARAJ	39	28	15	0	0	1	2	1	0	0
44	SELVAVATHY	41	28	15	0	0	0	1	0	0	0
45	VIJAY	45	27	15	0	0	1	2	0	0	0
46	LAKSHMANAN	49	29	15	0	1	1	3	1	0	0
47	SELVAM	45	26	15	0	0	0	1	0	0	0
48	SASIKUMAR	39	27	15	0	0	1	2	0	0	0
49	SELVARAJ	51	33	10	0	1	1	4	0	0	1
50	GOVINDHARAJ	48	29	15	0	0	1	2	0	0	0
51	SHANTHI	35	28	15	0	0	0	1	0	0	0
52	MOORTHY	48	31	15	0	1	1	3	1	0	0
53	POONKODI	50	26	15	0	0	1	2	0	0	0
54	SIVAKUMAR	45	27	15	0	0	0	1	1	0	0
55	SIVARAJ	44	29	15	0	0	1	2	0	0	0
56	ANTONY LEO	40	28	15	0	0	0	1	0	0	0
57	SARAVANA KU	34	30	15	0	0	1	2	0	0	0
58	KATHARARAYAN45	40	22	15	0	0	0	0	0	0	0
59	GANESH	39	30	9	0	0	1	3	1	0	0
60	GOVINDHARAJ	35	28	15	0	0	0	1	0	1	0
61	MURAGAN	40	22	15	0	0	0	0	0	0	0
62	ARUKANIYAMAL	35	30	15	0	0	0	1	0	0	1
63	ALLGAPAN	38	29	15	0	0	1	2	0	0	0
64	KAMARAJ	62	24	0	1	0	0	1	0	0	0
65	KATTHIRVEL	41	27	15	0	0	1	2	0	0	0
66	SIVAKUMAR	51	22	15	0	0	0	0	0	0	0
67	SELVARAJ	32	31	5	0	1	1	4	1	1	0
68	AL BATHA	48	26	15	0	0	0	1	0	0	0
69	SABUTHEEN	45	29	15	0	0	1	2	0	0	1
70	MANI	42	30	15	0	0	0	1	0	0	0
71	RANI	46	33	15	0	0	1	2	1	0	1
72	MURUGESH	43	19	15	0	0	0	0	0	0	0
73	SRINIVASAN	26	22	15	0	0	0	0	0	0	0
74	SANMUGASUNDRAM	34	30	15	0	0	0	1	0	0	0
75	KUPPURAJ	47	33	5	0	1	1	4	1	0	1
76	MURUGAIH	21	29	15	0	0	1	2	0	0	0
77	BALAMURUGAN	35	27	15	0	0	1	1	1	1	0
78	PRABU	23	28	15	0	0	1	2	0	0	0

79	RAJENDRAN	47	32	15	0	1	1	3	0	0	0
80	SELVAKUMAR	54	24	15	0	0	0	0	0	0	0
81	PARAMASIVA	18	27	15	0	0	1	2	1	0	0
82	UDYAKUMAR	36	28	15	0	0	1	2	1	0	0
83	NIRMALDEVAN	49	21	15	0	0	0	0	0	0	0
84	CHANDRAN	36	28	15	0	0	1	2	0	0	0
85	SUBBAIYA	33	26	15	0	0	0	1	0	0	1
86	ABDULKATAR	53	33	15	0	1	1	3	1	0	0
87	YESIN	42	31	15	0	0	1	2	0	0	0
88	JEGANATHAN	42	27	15	0	0	0	1	0	0	0
89	MANIKANDAN	44	21	15	0	0	1	0	0	0	0
90	ELANGOVAN	48	26	15	0	0	1	2	0	0	0
91	RANJENDRAN	20	19	15	0	0	0	0	0	0	0
92	KANAGARAJ	45	28	15	0	0	0	1	0	0	0
93	NAGARAJAN	72	31	15	1	0	1	2	0	0	0
94	RAMESH	40	28	15	0	0	0	1	0	0	0
95	ARJUNAN	20	26	15	0	0	1	4	1	0	0
96	KANNAPAN	45	29	15	0	0	1	2	0	0	0
97	MOHANRAJ	32	21	15	0	0	0	0	0	0	0
98	KANDSSAMY	45	31	15	0	0	1	2	0	0	1
99	RANGARAJ	21	19	15	0	0	0	0	0	0	0
100	NANTHAKUMAR	56	29	15	0	0	1	2	0	0	0
101	ROBERT	26	44	15	0	0	1	2	1	1	1
102	PONNAIYAN	64	31	15	1	0	0	1	0	0	0
103	GANESAN	32	34	10	0	1	1	4	0	0	0
104	VAIYAPURI	18	28	15	0	0	1	2	0	0	0
105	BALAKRISHNAN	40	30	15	0	0	0	1	1	0	0
106	ARIVALAGAN	28	27	15	0	0	1	2	0	0	0
107	VASANTHAN	32	18	15	0	0	0	0	0	0	0
108	KANTHARAJ	53	26	15	0	0	1	2	0	0	0
109	RAVICHANDRAN	37	29	15	0	0	1	2	0	0	0
110	SARAGAM	24	30	15	0	0	1	2	0	0	0
111	VELAVANTHAN	48	29	15	0	0	0	1	0	0	0
112	SENTHILVEL	47	28	15	0	0	1	2	0	0	0
113	VINAYAK	31	32	10	0	1	1	4	1	0	1
114	ULAGANATHAN	35	30	15	0	0	1	2	0	0	0
115	NARAYANAN	42	29	15	0	0	0	1	0	0	0
116	SANKARAN	38	29	15	0	0	1	2	1	0	0
117	JABAKUMAR	45	21	15	0	0	0	0	0	0	0

CTSI VALUES

Sl.no.	Name	Age	CT SCAN REPORT	CTSI	Panc Nec
1	VIJAY	37	B	2	0
2	MATHEWS	63	D	5	1
3	SIVA	16	B	2	0
4	KULANTHAISAMY	43	C	4	0
5	GOWRISANKER	23	A	1	0
6	RAMESHKUMAR	25	C	4	0
7	HUSSAIN	38	B	2	0
8	CHINNAN	53	A	1	0
9	KARUPPUSAMY	48	B	2	0
10	PRAKASH	32	A	1	0
11	KARUPPUSAMY	44	C	4	0
12	KARTHIKAYEN	65	C	4	0
13	MURUGESAN	23	B	2	1
14	ARUMUGAM	62	A	1	0
15	SENTHIL KUMAR	65	C	4	0
16	KRISHNAN	25	B	2	0
17	MUTHUKUMARSAY	48	C	4	0
18	JOHN BOSCO	20	B	2	0
19	SENTHIL KUMAR	22	D	7	1
20	SIDDIQUE	28	A	1	0
21	MOHAMAD	39	A	1	0
22	GOWRISANKER	37	D	5	1
23	RAMESH	33	B	2	0
24	BAKER	44	B	2	0
25	GANESH	26	C	4	0
26	SURESH	42	D	5	1
27	ALAGAPPAN	45	B	2	0
28	RAMALINGAM	39	B	2	0
29	SENTHIL VEL	22	A	1	0
30	BANNARI	34	B	2	0
31	KANNAN	44	C	4	0
32	THANGARAJ	27	A	1	0
33	DURAIRAJ	32	C	4	0
34	ARUMUGAM	23	B	2	0
35	KARRUPUSAMY	52	B	2	0
36	VELUSAMY	38	A	1	0
37	CHANDRAKALA	23	D	5	1
38	RAKIYYAPAN	37	B	2	0
39	SARAGANAN	39	C	4	0
40	UHAITHULA	39	B	2	0
41	VELAVANTHAN	50	C	4	0
42	SUBRAMANI	45	D	5	1

43	AROKIARAJ	39	B	2	0
44	SELVAVATHY	41	B	2	0
45	VIJAY	45	A	1	0
46	LAKSHMANAN	49	B	2	0
47	SELVAM	45	A	1	0
48	SASIKUMAR	39	B	2	0
49	SELVARAJ	51	D	7	1
50	GOVINDHARAJ	48	B	2	0
51	SHANTHI	35	C	4	0
52	MOORTHY	48	B	2	0
53	POONKODI	50	C	4	0
54	SIVAKUMAR	45	A	1	0
55	SIVARAJ	44	B	2	0
56	ANTONY LEO	40	A	1	0
57	SARAVANA KU	34	C	4	0
58	KATHARARAYAN45	40	B	2	0
59	GANESH	39	B	2	0
60	GOVINDHARAJ	35	D	5	0
61	MURAGAN	40	A	1	0
62	ARUKANIYAMAL	35	C	4	1
63	ALLGAPAN	38	B	2	0
64	KAMARAJ	62	A	1	0
65	KATTHIRVEL	41	B	2	0
66	SIVAKUMAR	51	B	2	0
67	SELVARAJ	32	A	1	0
68	AL BATHA	48	B	2	0
69	SABUTHEEN	45	D	5	1
70	MANI	42	A	1	0
71	RANI	46	D	5	1
72	MURUGESH	43	B	2	0
73	SRINIVASAN	26	B	2	0
74	SANMUGASUNDRAM	34	B	2	0
75	KUPPURAJ	47	D	5	1
76	MURUGAIH	21	A	1	0
77	BALAMURUGAN	35	B	2	0
78	PRABU	23	C	4	0
79	RAJENDRAN	47	A	1	0
80	SELVAKUMAR	54	C	4	0
81	PARAMASIVA	18	B	2	0
82	UDYAKUMAR	36	B	2	0
83	NIRMALDEVAN	49	A	1	0
84	CHANDRAN	36	B	2	0
85	SUBBAIYA	33	D	5	1
86	ABDULKATAR	53	C	4	0

87	YESIN	42	A	1	0
88	JEGANATHAN	42	C	4	0
89	MANIKANDAN	44	B	2	0
90	ELANGOVAN	48	A	1	0
91	RANJENDRAN	20	B	2	0
92	KANAGARAJ	45	C	4	0
93	NAGARAJAN	72	B	2	0
94	RAMESH	40	A	1	0
95	ARJUNAN	20	C	4	0
96	KANNAPAN	45	B	2	0
97	MOHANRAJ	32	A	1	0
98	KANDSSAMY	45	D	5	1
99	RANGARAJ	21	B	2	0
100	NANTHAKUMAR	56	B	2	0
101	ROBERT	26	D	5	1
102	PONNAIYAN	64	A	1	0
103	GANESAN	32	C	4	0
104	VAIYAPURI	18	B	2	0
105	BALAKRISHNAN	40	B	2	0
106	ARIVALAGAN	28	A	1	0
107	VASANTHAN	32	C	4	0
108	KANTHARAJ	53	B	2	0
109	RAVICHANDRAN	37	B	2	0
110	SARAGAM	24	C	4	0
111	VELAVANTHAN	48	B	2	0
112	SENTHILVEL	47	A	1	0
113	VINAYAK	31	B	2	1
114	ULAGANATHAN	35	A	1	0
115	NARAYANAN	42	C	4	0
116	SANKARAN	38	B	2	0
117	JABAKUMAR	45	A	1	0

A- CTSI SCORE- 1 , CT REPORT- ACUTE EDEMATOUS PANCREATITIS

B- CTSI SCORE-2 , CT REPORT-PANCREATIC GLAND ABNORMALITY AND PERIPANCREATIC INFLAMATIOM

C- CTSI SCORE -4 , C TREPORT-EXTRA PANCREATIC NECROSIS WITH FLUID COLLECTIONS

D- CTSI SCORE - > 4 CT REPORT- NECROTIC PANCREATITS

E-

F- RANSON SCORE MASTER CHART

Sl.no.	Name	Age	AGE	WBC	BLS	AST	ALT	HB	BUN	BASE DEFI	FLUID REPL	PaO2	CAL	Ranson	SAP	Etio	Morta
1	VIJAY	37	0	0	1	0	0	0	0	0	1	0	0	2	0	U1	0
2	MATHEWS	63	1	1	0	0	0	0	1	0	1	0	1	5	1	A	1
3	SIVA	16	0	1	1	0	0	0	0	0	0	0	0	2	0	A	0
4	KULANTHAISAMY	43	0	0	1	0	0	0	0	0	1	0	0	2	0	A	0
5	GOWRISANKER	23	0	0	0	0	0	0	0	0	1	0	0	1	1	U2	1
6	RAMESHKUMAR	25	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
7	HUSSAIN	38	0	0	0	0	0	0	0	0	1	0	0	1	0	A	0
8	CHINNAN	53	0	1	0	0	0	0	0	0	0	0	0	1	0	A	0
9	KARUPPUSAMY	48	0	1	1	0	0	0	0	0	0	0	0	2	0	U3	0
10	PRAKASH	32	0	1	0	0	0	1	1	1	1	0	0	5	1	A	0
11	KARUPPUSAMY	44	0	0	0	0	0	0	0	0	1	0	0	1	0	A	1
12	KARTHIKAYEN	65	1	0	1	0	0	0	0	0	1	0	0	3	0	U4	0
13	MURUGESAN	23	0	1	1	1	1	0	0	1	1	1	0	6	1	A	0
14	ARUMUGAM	62	1	0	0	0	0	0	0	0	1	0	0	2	0	G	0
15	SENTHIL KUMAR	65	1	0	0	0	0	0	0	0	1	0	0	2	0	A	0
16	KRISHNAN	25	0	0	0	0	0	0	0	0	1	0	0	1	0	U5	0
17	MUTHUKUMARSAY	48	0	1	0	0	0	0	0	0	1	0	0	2	0	A	0
18	JOHN BOSCO	20	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
19	SENTHIL KUMAR	22	0	0	0	0	0	0	1	0	1	0	0	2	1	U6	0
20	SIDDIQUE	28	0	0	1	0	0	0	0	0	1	0	0	2	0	A	0
21	MOHAMAD	39	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
22	GOWRISANKER	37	0	0	0	0	0	0	0	0	1	0	1	2	0	A	0
23	RAMESH	33	0	1	0	0	0	0	0	0	1	0	0	2	0	U7	0
24	BAKER	44	0	0	0	0	0	0	0	0	1	0	0	1	0	A	0
25	GANESH	26	0	1	1	0	0	0	0	0	0	0	0	2	0	A	0

26	SURESH	42	0	1	0	o	0	1	1	0	1	0	1	5	1	A	0
27	ALAGAPPAN	45	0	1	0	o	0	0	0	0	1	0	0	2	0	U8	0
28	RAMALINGAM	39	0	0	1	0	0	0	0	0	0	0	0	1	0	A	0
29	SENTHIL VEL	22	0	0	1	0	0	0	0	0	1	0	0	2	0	U9	0
30	BANNARI	34	0	1	0	0	0	1	0	1	1	0	0	4	1	A	0
31	KANNAN	44	0	1	0	0	0	0	0	0	0	0	0	1	0	A	0
32	THANGARAJ	27	0	1	0	0	0	0	0	0	1	0	0	2	0	A	0
33	DURAIRAJ	32	0	1	0	0	0	0	0	0	1	0	0	2	0	U10	0
34	ARUMUGAM	23	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
35	KARRUPUSAMY	52	0	1	0	0	0	0	0	0	1	0	0	2	0	A	0
36	VELUSAMY	38	0	1	1	0	0	1	1	1	1	0	0	6	1	A	1
37	CHANDRAKALA	23	0	0	0	0	0	0	0	0	1	0	0	1	0	U11	0
38	RAKIYYAPAN	37	0	1	1	0	0	0	0	0	0	0	0	2	0	A	0
39	SARAGANAN	39	0	0	0	0	0	0	0	0	1	0	0	1	0	A	0
40	UHAITHULA	39	0	0	0	0	0	0	0	0	1	0	0	1	0	U12	0
41	VELAVANTHAN	50	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
42	SUBRAMANI	45	0	1	1	0	0	0	0	0	1	0	0	3	0	U13	0
43	AROKIARAJ	39	0	0	0	0	0	0	1	0	1	0	0	2	1	A	0
44	SELVAVATHY	41	0	1	1	0	0	0	0	0	1	0	0	3	0	G	0
45	VIJAY	45	0	1	1	1	0	0	0	0	1	0	0	4	0	A	0
46	LAKSHMANAN	49	0	0	0	00	0	0	1	0	1	0	0	2	1	A	0
47	SELVAM	45	0	0	0	0	0	0	0	0	1	0	0	1	0	A	0
48	SASIKUMAR	39	0	1	1	0	0	0	0	0	0	0	0	2	0	A	0
49	SELVARAJ	51	0	1	1	1	1	0	0	0	1	0	0	5	0	U14	0
50	GOVINDHARAJ	48	0	1	1	0	0	0	0	0	0	0	0	2	0	0	0
51	SHANTHI	35	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
52	MOORTHY	48	0	0	0	0	0	0	1	0	1	0	0	2	1	A	0
53	POONKODI	50	0	0	1	0	0	0	0	0	0	0	0	1	0	G	0
54	SIVAKUMAR	45	0	1	0	0	0	1	1	1	1	0	1	6	1	U15	0
55	SIVARAJ	44	0	0	1	0	0	0	0	0	1	0	0	2	0	A	0

56	ANTONY LEO	40	0	0	1	0	0	0	0	0	0	0	0	1	0	A	0
57	SARAVANA KU	34	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
58	KATHARARAYAN45	40	0	0	1	0	0	0	0	0	1	0	0	2	0	U16	0
59	GANESH	39	0	0	1	0	0	0	0	0	0	0	0	1	1	A	0
60	GOVINDHARAJ	35	0	1	0	0	0	0	0	0	1	0	0	2	0	A	0
61	MURAGAN	40	0	1	1	0	0	0	0	0	1	0	0	3	0	AA	0
62	ARUKANIYAMAL	35	0	0	0	0	0	0	0	0	1	0	0	1	0	U17	0
63	ALLGAPAN	38	0	1	1	0	0	0	0	0	0	0	0	2	0	A	0
64	KAMARAJ	62	1	0	0	0	0	0	0	0	0	0	0	1	0	A	0
65	KATTHIRVEL	41	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
66	SIVAKUMAR	51	0	0	1	0	0	0	0	0	0	0	0	1	0	U18	0
67	SELVARAJ	32	0	1	1	0	0	1	1	0	1	1	0	6	1	A	1
68	AL BATHA	48	0	0	1	0	0	0	0	0	1	0	0	2	0	A	0
69	SABUTHEEN	45	0	0	1	0	0	0	0	0	0	0	0	1	0	A	0
70	MANI	42	0	1	1	0	0	0	0	0	0	0	0	2	0	A	0
71	RANI	46	0	0	0	0	0	0	1	0	1	0	0	2	1	A	0
72	MURUGESH	43	0	1	1	0	0	0	0	0	0	0	0	2	0	A	0
73	SRINIVASAN	26	0	1	1	0	0	0	0	0	1	0	0	3	0	U19	0
74	SANMUGASUNDRAM	34	0	1	0	0	0	0	0	0	1	0	0	2	0	A	0
75	KUPPURAJ	47	0	0	1	0	0	0	0	0	1	0	0	2	1	A	0
76	MURUGAIH	21	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
77	BALAMURUGAN	35	0	0	0	0	0	0	1	0	1	0	0	2	1	U20	0
78	PRABU	23	0	0	1	0	0	0	0	0	1	0	0	2	0	A	0
79	RAJENDRAN	47	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
80	SELVAKUMAR	54	0	0	0	0	0	0	0	0	1	0	0	1	0	A	0
81	PARAMASIVA	18	0	0	0	0	0	0	1	0	1	0	0	2	1	U21	0
82	UDYAKUMAR	36	0	1	1	0	0	1	1	1	1	0	0	6	1	A	0
83	NIRMALDEVAN	49	0	1	0	0	0	0	0	0	1	0	0	2	0	A	0

84	CHANDRAN	36	0	0	1	0	0	0	0	0	0	0	0	1	0	A	0
85	SUBBAIYA	33	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
86	ABDULKATAR	53	0	0	0	0	0	0	1	0	1	0	0	2	1	A	0
87	YESIN	42	0	0	1	0	0	0	0	0	0	0	0	1	0	A	0
88	JEGANATHAN	42	0	1	1	0	0	0	0	0	1	0	0	3	0	U22	0
89	MANIKANDAN	44	0	0	1	0	0	0	0	0	1	0	0	2	0	A	0
90	ELANGOVAN	48	0	1	0	0	0	0	0	0	1	0	0	2	0	A	0
91	RANJENDRAN	20	0	0	1	0	0	0	0	0	1	0	0	2	0	A	0
92	KANAGARAJ	45	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
93	NAGARAJAN	72	1	0	1	0	0	0	0	0	0	0	0	2	0	U23	0
94	RAMESH	40	0	0	1	0	0	0	0	0	0	0	0	1	0	A	0
95	ARJUNAN	20	0	1	1	0	0	0	1	1	1	0	1	6	1	A	0
96	KANNAPAN	45	0	1	0	0	0	0	0	0	1	0	0	2	0	A	0
97	MOHANRAJ	32	0	0	1	0	0	0	0	0	0	0	0	1	0	A	0
98	KANDSSAMY	45	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
99	RANGARAJ	21	0	0	1	0	0	0	0	0	1	0	0	2	0	A	0
100	NANTHAKUMAR	56	0	1		0	0	0	0	0	1	0	0	2	0	A	0
101	ROBERT	26	0	1	1	0	0	0	1	1	1	1	0	6	1	A	1
102	PONNAIYAN	64	1	0	0	0	0	0	0	0	0	0	0	1	0	A	0
103	GANESAN	32	0	0	1	0	0	0	0	0	1	0	0	2	0	A	0
104	VAIYAPURI	18	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
105	BALAKRISHNAN	40	0	1	0	0	0	0	0	0	1	0	0	2	1	A	0
106	ARIVALAGAN	28	0	1	0	0	0	0	0	0	1	0	0	2	0	A	0
107	VASANTHAN	32	0	0	1	0	0	0	0	0	1	0	0	2	0	G	0
108	KANTHARAJ	53	0	0	0	0	0	0	0	0	1	0	0	1	0	A	0
109	RAVICHANDRAN	37	0	1	0	0	0	0	0	0	1	0	0	2	0	A	0
110	SARAGAM	24	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
111	VELAVANTHAN	48	0	0	1	0	0	0	0	0	0	0	0	1	0	A	0
112	SENTHILVEL	47	0	1	1	0	0	0	0	0	1	0	0	3	0	A	0
113	VINAYAK	31	0	0	1	0	0	0	0	0	1	0	0	2	1	A	1

114	ULAGANATHAN	35	0	0	0	0	0	0	0	0	1	0	0	1	0	A	0
115	NARAYANAN	42	0	0	0	1	1	0	0	0	1	0	0	3	0	A	0
116	SANKARAN	38	0	1	0	0	0	0	0	0	1	0	0	2	1	A	0
117	JABAKUMAR	45	0	0	1	0	0	0	0	0	1	0	0	2	0	A	0

G-

H-

I-

J-

Age (yr)	1->55	>70*
WBC (×1000/mm3)1-	1->16	>18
Glucose (mg/dl)	1->200	>220
AST (IU/L)	1->250	>250
LDH (IU/L)	1->350	>400

Within 48 Hours of Admission

Hematocrit decrease (points)	1->10	>10
BUN increase (mg/dl)	1->5	>2
Deficit in base(mEq/L)	1->4	>5
Fluid replaced (L)	1->6	>4
Pa_{O_2} (mm Hg)	1-<60	<60
Calcium (mg/dl)	1-<8	<8

K- * value for biliary acute pancreatitis
L- Value for 1 in the master chart is given above other than that value is entered as 0
M-

ON THE WHOLE MASTER CHART FOR ALL SCORE

Sl.no	Name	Age	Sex	IP No	Ranson	BISAP	CTSI	SAP	Panc Nec	Mortal	Morbi	Etiology
1	VIJAY	37	M	33208	2	2	2	0	0	0	8	U1
2	MATHEWS	63	M	26065	5	4	5	1	1	1	10	A
3	SIVA	16	M	25125	2	1	2	0	0	0	10	A
4	KULANTHAISAMY	43	M	21060	2	0	4	0	0	0	5	A
5	GOWRISANKER	23	M	31785	1	3	1	1	0	1	4	U2
6	RAMESHKUMAR	25	M	31158	3	2	4	0	0	0	5	A
7	HUSSAIN	38	M	22379	1	2	2	0	0	0	5	A
8	CHINNAN	53	M	20870	1	1	1	0	0	0	8	A
9	KARUPPUSAMY	48	M	35280	2	2	2	0	0	0	4	U3
10	PRAKASH	32	M	34852	5	3	1	1	0	0	13	A
11	KARUPPUSAMY	44	M	33135	1	2	4	0	0	1	7	A
12	KARTHIKAYEN	65	M	67892	3	2	4	0	0	0	10	U4
13	MURUGESAN	23	M	22838	6	4	2	1	1	0	2	A
14	ARUMUGAM	62	M	2043	2	1	1	0	0	0	3	G

				0								
15	SENTHIL KUMAR	65	M	13165	2	5	4	0	0	0	8	A
16	KRISHNAN	25	M	9190	1	0	2	0	0	0	5	U5
17	MUTHUKUMARSAY	48	M	5196	2	2	4	0	0	0	5	A
18	JOHN BOSCO	20	M	19428	3	2	2	0	0	0	12	A
19	SENTHIL KUMAR	22	M	35865	2	1	7	1	1	0	15	U6
20	SIDDIQUE	28	M	59190	2	2	1	0	0	0	3	A
21	MOHAMAD	39	M	60001	3	1	1	0	0	0	3	A
22	GOWRISANKER	37	M	49892	2	1	5	0	1	0	6	A
23	RAMESH	33	M	60013	2	2	2	0	0	0	2	U7
24	BAKER	44	M	60622	1	0	2	0	0	0	5	A
25	GANESH	26	M	41640	2	3	4	0	0	0	7	A
26	SURESH	42	M	39597	5	2	5	1	1	0	6	A
27	ALAGAPPAN	45	M	41811	2	2	2	0	0	0	5	U8
28	RAMALINGAM	39	M	43832	1	2	2	0	0	0	3	A
29	SENTHIL VEL	22	M	10813	2	1	1	0	0	0	4	U9
30	BANNARI	34	M	1021	4	3	2	1	0	0	10	A

				1								
31	KANNAN	44	M	10813	1	1	4	0	0	0	3	A
32	THANGARAJ	27	M	6113	2	0	1	0	0	0	2	A
33	DURAIRAJ	32	M	1759	2	2	4	0	0	0	5	U10
34	ARUMUGAM	23	M	3864	3	2	2	0	0	0	4	A
35	KARRUPUSAMY	52	M	3916	2	1	2	0	0	0	3	A
36	VELUSAMY	38	M	6012	6	4	1	1	0	1	10	A
37	CHANDRAKALA	23	F	42390	1	2	5	0	1	0	5	U11
38	RAKIYYAPAN	37	M	33207	2	1	2	0	0	0	3	A
39	SARAGANAN	39	M	40428	1	2	4	0	0	0	3	A
40	UHAITHULA	39	M	40285	1	0	2	0	0	0	2	U12
41	VELAVANTHAN	50	M	41292	3	2	4	0	0	0	2	A
42	SUBRAMANI	45	M	58804	3	4	5	0	1	0	8	U13
43	AROKIARAJ	39	M	36940	2	2	2	1	0	0	3	A
44	SELVAVATHY	41	F	59486	3	1	2	0	0	0	6	G
45	VIJAY	45	M	60834	4	2	1	0	0	0	5	A
46	LAKSHMANAN	49	M	60828	2	3	2	1	0	0	7	A
47	SELVAM	45	F	61084	1	1	1	0	0	0	8	A

48	SASIKUMAR	39	M	61666	2	2	2	0	0	0	1	A
49	SELVARAJ	51	M	61746	5	4	7	0	1	0	6	U14
50	GOVINDHARAJ	48	M	62733	2	2	2	0	0	0	5	A
51	SHANTHI	35	F	62920	3	1	4	0	0	0	3	A
52	MOORTHY	48	M	61400	2	3	2	1	0	0	8	A
53	POONKODI	50	F	63329	1	2	4	0	0	0	7	G
54	SIVAKUMAR	45	M	63523	6	1	1	1	0	0	2	U15
55	SIVARAJ	44	M	63892	2	2	2	0	0	0	4	A
56	ANTONY LEO	40	M	73866	1	1	1	0	0	0	2	A
57	SARAVANA KU	34	M	67043	3	2	4	0	0	0	8	A
58	KATHARARAYAN45	40	M	64827	2	0	2	0	0	0	3	U16
59	GANESH	39	M	59834	1	3	2	1	0	0	8	A
60	GOVINDHARAJ	35	M	59060	2	1	5	0	0	0	9	A
61	MURAGAN	40	M	70798	3	0	1	0	0	0	2	AA
62	ARUKANIYAMAL	35	F	58157	1	1	4	0	1	0	10	U17

63	ALLGAPAN	38	M	66373	2	2	2	0	0	0	7	A
64	KAMARAJ	62	M	65078	1	1	1	0	0	0	4	A
65	KATTHIRVEL	41	M	65078	3	2	2	0	0	0	7	A
66	SIVAKUMAR	51	M	60008	1	0	2	0	0	0	6	U18
67	SELVARAJ	32	M	58753	6	4	1	1	0	1	5	A
68	AL BATHA	48	M	71454	2	1	2	0	0	0	8	A
69	SABUTHEEN	45	M	71341	1	2	5	0	1	0	3	A
70	MANI	42	M	53912	2	1	1	0	0	0	6	A
71	RANI	46	F	16007	2	2	5	1	1	0	7	A
72	MURUGESH	43	M	48884	2	0	2	0	0	0	8	A
73	SRINIVASAN	26	M	47612	3	0	2	0	0	0	4	U19
74	SANMUGASUNDRAM	34	M	46250	2	1	2	0	0	0	8	A
75	KUPPURAJ	47	M	44122	2	4	5	1	1	0	8	A
76	MURUGAIH	21	M	69416	3	2	1	0	0	0	12	A
77	BALAMURUGAN	35	M	50522	2	1	2	1	0	0	2	U20

78	PRABU	23	M	66706	2	2	4	0	0	0	4	A
79	RAJENDRAN	47	M	52111	3	3	1	0	0	0	5	A
80	SELVAKUMAR	54	M	48914	1	0	4	0	0	0	4	A
81	PARAMASIVA	18	M	47322	2	2	2	1	0	0	3	U21
82	UDYAKUMAR	36	M	71470	6	2	2	1	0	0	5	A
83	NIRMALDEVAN	49	M	33787	2	0	1	0	0	0	8	A
84	CHANDRAN	36	M	33800	1	2	2	0	0	0	6	A
85	SUBBAIYA	33	M	36916	3	1	5	0	1	0	15	A
86	ABDULKATAR	53	M	38342	2	3	4	1	0	0	2	A
87	YESIN	42	M	38459	1	2	1	0	0	0	6	A
88	JEGANATHAN	42	M	40252	3	1	4	0	0	0	3	U22
89	MANIKANDAN	44	M	41360	2	0	2	0	0	0	5	A
90	ELANGOVAN	48	M	42799	2	2	1	0	0	0	10	A
91	RANJENDRAN	20	M	45075	2	0	2	0	0	0	5	A
92	KANAGARAJ	45	M	52930	3	1	4	0	0	0	4	A

93	NAGARAJAN	72	M	60361	2	2	2	0	0	0	5	U23
94	RAMESH	40	M	58934	1	1	1	0	0	0	8	A
95	ARJUNAN	20	M	71987	6	4	4	1	0	0	13	A
96	KANNAPAN	45	M	70812	2	2	2	0	0	0	12	A
97	MOHANRAJ	32	M	70812	1	0	1	0	0	0	6	A
98	KANDSSAMY	45	M	61754	3	2	5	0	1	0	6	A
99	RANGARAJ	21	M	54298	2	0	2	0	0	0	5	A
100	NANTHAKUMAR	56	M	39524	2	2	2	0	0	0	5	A
101	ROBERT	26	M	17552	6	2	5	1	1	1	5	A
102	PONNAIYAN	64	M	16797	1	1	1	0	0	0	2	A
103	GANESAN	32	M	12523	2	4	4	0	0	0	5	A
104	VAIYAPURI	18	M	14460	3	2	2	0	0	0	5	A
105	BALAKRISHNAN	40	M	14294	2	1	2	1	0	0	15	A
106	ARIVALAGAN	28	M	14335	2	2	1	0	0	0	10	A
107	VASANTHAN	32	M	13077	2	0	4	0	0	0	6	G

108	KANTHARAJ	53	M	11595	1	2	2	0	0	0	15	A
109	RAVICHANDRAN	37	M	65976	2	2	2	0	0	0	10	A
110	SARAGAM	24	M	68435	3	2	4	0	0	0	6	A
111	VELAVANTHAN	48	M	63555	1	1	2	0	0	0	20	A
112	SENTHILVEL	47	M	63647	3	2	1	0	0	0	11	A
113	VINAYAK	31	M	48837	2	4	2	1	1	1	1	A
114	ULAGANATHAN	35	M	40252	1	2	1	0	0	0	13	A
115	NARAYANAN	42	M	38489	3	1	4	0	0	0	7	A
116	SANKARAN	38	M	48401	2	2	2	1	0	0	8	A
117	JABAKUMAR	45	M	45684	2	0	1	0	0	0	4	A

A – Alcohol
U – Unknown
G- Galbadder

PATIENT CONSENT FORM

STUDY: A COMPARATIVE STUDY OF VARIOUS PROGNOSTICS SCORING SYSTEM (RANSON, BISAP, CTSI) IN ACUTE PANCREATITIS

This study has been explained to me in my own language and I understood the following

1. What the study involves
2. That the refusal to participate will not affect my treatment in any way
3. That I may withdraw to take part in this study

Signature of the patient:

Full name of the patient:

Address:

Date:

Witness: (should be a person not connected with the study)

I have been present while the procedure to be performed has been explained to the patient and I have witnessed his/her consent to take part.

Signature of the witness:

Full name of the witness:

Address:

Date: